"Few people are capable of expressing with equanimity opinions which differ from the prejudices of their social environment. Most people are even incapable of forming such opinions."

—Albert Einstein

INSIDE

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

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

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1900 L Street NW, Suite 725
Washington, DC 20036
202-833-8850
E-mail: enc@enc-online.org

NUTRITION CLOSE-UP

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Both High- and Low-Carbohydrate Diets Confer Unique Benefits in Treating the Metabolic Syndrome

The metabolic syndrome—a condition characterized by central obesity, dyslipidemia, hypertension, and insulin resistance—has become all too common, growing in tandem with obesity rates. This condition affects nearly one quarter of the adult population in the US and doubles an individual's risk for cardiovascular disease. Although weight loss is generally considered first-line therapy, the optimal diet for treating the metabolic syndrome and its comorbidities has not been identified. Hypocaloric low-fat, high-carbohydrate diets and moderate-fat, higher-protein diets have both been shown to induce weight loss—an important outcome for this population—but until recently, their relative effectiveness in treating the metabolic syndrome had not been tested.

In a randomized, prospective study, Muzio et al. compared the effects of two hypocaloric diets differing in macronutrient composition on cardiovascular disease risk factors in 100 obese patients with the metabolic syndrome. The participants (27 men, 73 women) were randomly assigned to one of two diets differing in carbohydrate and protein content. The end weight-loss goal was a 5% decrease in body weight from baseline. Both groups were similar with respect to physical characteristics and the presence of metabolic syndrome risk components at baseline.

Many of the improvements observed in these study participants…were directly associated with weight loss. However, the differences in macronutrient [intake]…provided benefits beyond those associated with a simple reduction in body weight.

The high-carbohydrate diet provided 65% of calories from carbohydrate, 13% from protein, and 22% from fat. The low-carbohydrate diet provided 48% of energy from carbohydrates, 19% from protein, and 33% from fat, with an emphasis on animal protein and monounsaturated fat. Both diets complied with the current dietary recommendations of the National Cholesterol Education Program Adult Treatment Panel III. Calorie requirements were calculated for each participant and individual prescribed diets were designed to provide a deficit of 500 kcals/day, a discrepancy that would result in a 5% decrease in body weight from baseline. Participants were also encouraged to increase their daily physical activity.

Body weight, height, BMI, waist circumference, blood pressure, and serum lipids were determined for each participant at baseline and at the conclusion of the 5-month trial. For the duration of the study, participants attended monthly group sessions to receive diet guidance. Adherence to the diet was monitored by a 20-item food intake questionnaire administered during the final group session, and was further evaluated by how close participants came to their goal weight.

Continued on page 2

Although only 5 of the 100 participants actually increased their physical activity over the course of the trial, all attended the group sessions and completed the study. Of the patients in the high-carbohydrate group, 92% achieved a weight loss of >5% of baseline body weight. Of those in the low-carbohydrate group, 84% met their goal weight. Both diet groups experienced improvements in body weight (10% decrease from baseline), BMI, waist circumference, prevalence of central obesity, systolic and diastolic blood pressures, total cholesterol, triacylglycerol concentrations, blood glucose, insulin, and measures of insulin resistance over the course of the study. HDL cholesterol levels did not change significantly in either group.

Beyond these improvements, however, each diet regimen conferred its own unique benefits. Participants following the low-carbohydrate diet saw greater decreases in systolic blood pressure and triacylglycerol concentrations, and experienced a reduction in heart rate that was not observed in the other participants. Likewise, decreases in LDL cholesterol concentrations were seen only in participants following the high-carbohydrate regimen.

Multiple regression analysis showed that protein was the only dietary component significantly associated with the changes in systolic blood pressure ($B=-0.0018$, $P<0.05$) and that carbohydrate intake ($B=-0.049$, $P<0.05$) and weight loss ($B=1.860$, $P<0.01$) were responsible for changes in serum triacylglycerol concentrations. By the conclusion of the study, 54% of the participants in the low-carbohydrate group and 40% of those in the high-carbohydrate group no longer met the diagnostic criteria for the metabolic syndrome (although the difference between groups was not significant).

Many of the improvements observed in these study participants—including improvements in blood pressure, metabolic abnormalities, total cholesterol, serum triacylglycerol, blood glucose, insulin, and measures of insulin resistance—were directly associated with weight loss. However, the differences in macronutrient composition between diets provided benefits beyond those associated with a simple reduction in body weight. The low-carbohydrate diet, rich in protein and moderate in fat content, reduced the prevalence of hypertension and hypertriacylglycerolemia and improved heart rates. Conversely, the high-carbohydrate diet resulted in greater improvements in LDL cholesterol. These observations suggest that both high- and low-carbohydrate diets are effective in improving abnormalities associated with the metabolic syndrome and that the preferential use of one diet regimen over the other might best be determined by the individual patient's metabolic profile. Those with high LDL cholesterol levels may benefit from a high-carbohydrate, hypocaloric regimen, while those with hypertension and hypertriglyceridemia might respond more favorably to a low-carbohydrate, hypocaloric diet pattern. ■

Muzio F, Mondazzi L, Harris W, et al. Effects of moderate variations in the macronutrient content of the diet on cardiovascular disease risk factors in obese patients with the metabolic syndrome. *Am J Clin Nutr* 2007;86:946-51.

KEY MESSAGES

- Both diet groups experienced improvements in body weight (10% decrease from baseline), BMI, waist circumference, prevalence of central obesity, systolic and diastolic blood pressures, total cholesterol, triacylglycerol concentrations, blood glucose, insulin, and measures of insulin resistance over the course of the study.
- By the conclusion of the study, 54% of the participants in the low-carbohydrate group and 40% of those in the high-carbohydrate group no longer met the diagnostic criteria for the metabolic syndrome (no significant difference between groups).
- Reductions in LDL cholesterol concentrations were seen only in participants following the high-carbohydrate regimen.
- Participants following the low-carbohydrate diet saw greater decreases in systolic blood pressure and triacylglycerol concentrations, and experienced a reduction in heart rate that was not observed in the other participants.

Mediterranean Diet Might be Especially Protective for Diabetic Adults

Modern medicine relies predominantly on established risk factors (such as dyslipidemia, excess body weight, hypertension, history of diabetes, existing CVD, etc…) to estimate an individual's likelihood of developing coronary heart disease (CHD). However, observations from a recent study conducted in Australia suggest that diet might play a more important role in modifying CHD risk than previously thought. The findings further suggest that certain components of the Mediterranean diet might prove protective against CHD even for individuals exhibiting these risk factors.

The study was initiated after researchers in Australia noted that despite the prevalence of traditional risk factors in this group, migrants from Mediterranean countries have lower CVD mortality rates than their native-born Australian counterparts. The traditional Mediterranean diet is characterized by frequent consumption of fish and plant foods, moderate wine intake, and infrequent consumption of meats. Foods traditionally included in this pattern, such as fish, olive oil, and legumes are thought to have antioxidant and anti-inflammatory constituents that might counterbalance other sources of CVD risk.

To evaluate the influence of the Mediterranean diet pattern on CVD mortality in Australia, the researchers analyzed food-frequency information collected during the prospective Melbourne Collaborative Cohort Study. The large study group (n=40,653; 23,980 women and 16,673 men) consisted of adult residents of Melbourne, Australia, aged 40-69 years at baseline. Participants from southern European countries were deliberately oversampled, comprising 24% of the study group.

Participants filled out a 121-item food frequency questionnaire at baseline to reflect consumption over the preceding year. Participants were also interviewed at baseline to obtain information on potential confounding factors including country of birth, smoking status, level of education, total daily energy intake, physical activity, history of CVD, family history of CVD, history of diabetes or hypertension, social isolation, waist-to-hip ratio, body mass index (BMI), and sex.

The researchers used factor analysis to identify 4 predominant dietary patterns from the intake records. The "Mediterranean" diet factor was characterized by frequent consumption of garlic, cucumber, olive oil, salad greens, peppers, cooked dried legumes, legume soups, feta and ricotta cheeses, olives, steamed fish, and boiled chicken. The "vegetable" factor was characterized by frequent intake of cauliflower, broccoli, carrots, cabbage or Brussels sprouts, pumpkin, green beans or peas, leafy greens, celery or fennel, potato cooked without fat, beetroot, zucchini/squash/ /eggplant, coleslaw, salad greens, cucumber, and peppers.

The "meat" factor was characterized by frequent intake of beef rissoles, roast beef or veal, fried potato, beef or veal schnitzel, savory pastries, mixed dishes with lamb, fried eggs, beef steaks, fried fish, and bacon. The "fresh fruit" factor was defined by frequent intake of apricots, peaches/nectarines, plums, cantaloupe/honeydew, grapes, watermelon, pears, strawberries, oranges/mandarins, figs, apples, and pineapple. The meat factor accounted for 33% of the variation in daily energy intake, with those in the highest quartile reporting the highest daily energy intakes.

An average follow-up period of 10.4 years yielded a total of 697 deaths from CVD (407 from ischemic heart disease). After adjustment for waist-to-hip ratio, BMI, history of diabetes, and hypertension, higher Mediterranean factor scores were associated with lower CVD and IHD death rates. The meat factor was not associated with CVD or IHD mortality.

Some intriguing findings emerged when the data were analyzed for specific populations within the study cohort. Participants with diabetes appeared to benefit more from the Mediterranean diet factor than their non-diabetic counterparts. The hazard ratios (HRs) for quartiles 2-4 (compared to quartile 1) were 0.42 (95% CI: 0.18, 0.97), 0.40 (95% CI: 0.17, 0.97), and 0.21 (95% CI: 0.09, 0.47) for participants with diabetes. For those without diabetes, the HR for the highest compared with the lowest quartile was 0.71; (95% CI: 0.47, 1.08). Participants with diabetes also exhibited a different response to the vegetable factor. Compared to quartile 1, the HRs for quartiles 2-4 were 2.32 (95% CI: 1.02, 5.26), 2.50 (95% CI: 1.07, 5.84), and 1.74 (95% CI: 0.71, 4.27), respectively. For participants with no history of CVD, (after adjustment for the aforementioned covariates) the vegetable and fresh fruit factors were both inversely associated with CVD death and the Mediterranean diet factor was inversely associated with both CVD and IHD mortality.

Weaknesses of the study model include reliance on food frequency questionnaires (and thus participants' memories) to obtain dietary information and restriction of dietary data to the 12 months preceding the study (which might not be representative of diet during the causative relevant period), among others.

The Mediterranean diet is characterized by frequent intake of foods that provide antioxidants and phytochemicals thought to be beneficial for human health. Perhaps just as important, adherence to this diet is also associated with infrequent consumption of foods such as cream, sour cream, ice cream, chocolate, sausages, jams, honey, cake, and sweet biscuits that are high in salt, saturated fats, and refined carbohydrates, and low in fiber. The authors postulate that this might partially explain why participants with diabetes were particularly responsive to the Mediterranean diet pattern, since the avoidance of these nutrients suggests better metabolic control.

This study provides valuable insight into the role of dietary factors (particularly those related to the Mediterranean diet) that might mitigate CVD risk among diabetic and non-diabetic populations. These observations suggest that the Mediterranean diet is protective against CVD mortality for adults in this population and that it might be especially beneficial for diabetic adults. ■

Harriss LR, English DR, Powles J, et al. Dietary patterns and cardiovascular mortality in the Melbourne Collaborative Cohort Study. *Am J Clin Nutr* 2007;86:221-229.

READ IT AGAIN... FOR THE FIRST TIME

1985

The Diet-Heart Question in 1985: Has it really been settled?

A pioneering researcher in lipid metabolism, E.H. "Pete" Ahrens, Jr. (1915-2000) was respected by his colleagues and widely-published in his field. Utilizing the emerging methodologies of his time, he was among the first to report the changes in blood cholesterol levels induced by modifying dietary fat intake. He was also among the first to recognize that a number of hereditary and lifestyle factors (besides diet) strongly influenced plasma lipids and that no single dietary hypothesis could explain the occurrence of heart disease. While highly-regarded by his colleagues, Ahrens' reputation did not stand in the way of his integrity as a researcher, nor his willingness to be part of the vocal minority when it came to matters of practical application of scientific research. "…Well known as a major contributor to the diet-serum lipid-atherosclerosis relationship, he objected to popular simplifications that would lay all coronary artery disease at the doorstep of diet" (1).

In December, 1984, the NIH held its Consensus Development Conference on Lowering Cholesterol to Prevent Heart Disease. At that time, coronary heart disease (CHD) had been identified as the cause of over 550,000 deaths per year in the US, and the correlation between CHD and elevated serum cholesterol levels contributed a real sense of urgency to the development of improved screening and treatment protocols. Thus, the matter of reducing serum cholesterol levels had become a top priority for those involved in public health policy. The two-day consensus conference convened for this purpose included cardiologists, primary care physicians, epidemiologists, biomedical scientists, lipoprotein experts, biostatisticians, experts in preventive medicine, and lay representatives. Their purpose was to come to a research-based consensus on the most effective means by which cholesterol levels could be reduced in the general US population and to provide recommendations to guide public policy in addressing this matter.

The dietary recommendations ultimately adopted by the panel were essentially the same as those delineated in the American Heart Association's (AHA) "prudent diet" (a regimen originally developed by the AHA to treat individuals at high risk for CHD). The prudent diet would limit total fat to no more than 30% of total calories (with saturated fat contributing no more than 10% and polyunsaturated oils contributing 10%), and cholesterol to 250-300 mg/day. These recommendations would apply to every man, woman, and child over age 2.

Skeptical of broad, sweeping untested recommendations that would potentially alter the dietary course of an entire population, Ahrens shared his misgivings in the pages of *The Lancet*, May 11, 1985 (2). Ahrens' perspective is interesting, at the very least, in light of today's complex nutrition issues and the sometimes difficult quandary of applying advancing scientific knowledge to public policy.

Three important questions were raised during the consensus conference: 1) whether the prudent diet would, indeed, reduce the incidence of CHD; 2) whether the diet was safe and effective for all subsets of the population over age 2; and 3) whether this diet was the best dietary regimen available to reduce blood cholesterol levels and CVD risk. In his submission to The Lancet, Ahrens presented an analysis of the "state of the science," the research upon which the panel's recommendations were ultimately based, and potential consequences of a broad application of such recommendations.

1) Would the "prudent diet" reduce the incidence of CHD?

"In countries with diets lower in [calories, saturated fat, and cholesterol], blood cholesterol levels are lower, and coronary heart disease is less common. There is no doubt that appropriate changes in our diet will reduce blood cholesterol levels. Epidemiologic data and over a dozen clinical trials allow us to predict with reasonable assurance that such a measure will afford significant protection against coronary heart disease" (3).

Ahrens felt that the panel's conclusions based on epidemiological data were overly optimistic. While epidemiological research provides valuable insight, "correlations, no matter how strong, are never proof…for instance, to what degree [were] the CHD rates of Irish migrants to Boston (or Japanese to Hawaii) due to changes in lifestyle or to economic and social changes, rather than (as now assumed) to dietary changes?" (2) Thus, even studies that strongly suggest an association between a variable and an endpoint cannot be used as proof that the two are causally related.

One of the studies most heavily relied upon by the consensus conference to justify its conclusions and recommendations was

the Lipid Research Clinic's coronary primary prevention trial (LRC-CPPT) (4). The LRC-CPPT was a drug trial in which cholestyramine was used to reduce plasma cholesterol levels in hypercholesterolemic men. It was one of 20 trials predating the consensus conference that had attempted to reduce plasma cholesterol by dietary or pharmacological means…and the only one to produce evidence that cholesterol lowering would lead to a reduction in CHD risk. (Note: Risk reduction was observed only in the men with cholesterol levels in the top 5% of the cholesterol distribution curve.) While pooled data from the remaining diet and drug trials did show a statistically significant improvement in CHD risk with reduced blood cholesterol levels, they also showed a disturbing (if not significant) increase in non-CHD related deaths associated with cholesterol lowering. Such a finding probably warranted further research before broad cholesterol lowering recommendations were adopted and publicized. Ahrens further stated that there was virtually no data to support the assumption that the prudent diet would be as effective in all segments of the population as the cholestyramine treatment had been in this subset of men, citing the difference in mechanisms between dietary and pharmacological treatments and the question of whether the same degree of cholesterol lowering by dietary means would lead to a similar reduction in CHD risk.

2) Would the diet be safe and effective for all people >age 2?

"It has been established beyond a reasonable doubt that lowering definitely elevated blood cholesterol levels…will reduce the risk of heart attacks due to coronary heart disease. This has been demonstrated most conclusively in men with elevated blood cholesterol levels, but much evidence justifies the conclusion that similar protection will be afforded in women with elevated levels" (3).

According to data from the Multiple Risk Factor Intervention Trial (MRFIT) (5), CHD risk does not increase linearly with plasma cholesterol levels. Instead, the line for risk is relatively flat until it reaches the highest cholesterol levels, where it rapidly slopes upward. Thus, the greatest benefit from cholesterol lowering occurs in those with the highest plasma cholesterol levels. Ahrens pointed out that most of the published research studies available at the time of the consensus conference had been conducted in male participants who were considered high-risk for CHD because of existing hypercholesterolemia. This group is the most likely to see benefits from a reduction in plasma cholesterol (and most likely to have the greatest number of CHD events during a given trial period), and is, therefore, the population subset most often selected for studies of this kind.

Basing his comments on the assumptions made by the consensus panel that the LRC-CPPT results could be generalized to an entire population, Ahrens wrote, "I seriously doubt that the benefits of cholesterol-lowering seen in the highest risk males can be expected to occur also in men, women, and children with lower plasma cholesterol levels, especially when a different and untested method of intervention—namely, the prudent diet—is applied" (2). Ahrens also disagreed with the assumption that the prudent diet would be safe and effective in all children over the age of 2, citing his concerns that there was no research to support the assumption that growth and development in these children would not be hampered by restricting total fat, saturated fat, or cholesterol intake.

3) Was the prudent diet the best regimen available to reduce blood cholesterol levels and CVD risk?

Lastly, Ahrens questioned the panel's choice of therapeutic dietary regimen to achieve cholesterol reduction goals (namely the "prudent diet," as set forth by the American Heart Association), citing evidence that several other dietary interventions could have been recommended with equal or stronger scientific support. For instance, a vegetarian diet with a high polyunsaturated to saturated fat ratio might have been equally effective. There was also evidence that diets high in monounsaturated fats were associated with low serum cholesterol levels and low rates of CHD. Still other clinical trials had shown that the substitution of polyunsaturated with monounsaturated fats could reduce total cholesterol levels without decreasing HDL concentrations. Diets including fish oils had also been associated with total cholesterol and triacylglycerol levels and with low rates of CHD. Ahrens also reminded readers that "there…is abundant evidence that the quality of fat ingested is a far stronger determinant of [plasma cholesterol levels] than is the amount of cholesterol ingested." Why, then, was the prudent diet chosen over all other potential candidates? Besides the evidence provided by two studies showing that a reduction in total fat would reduce serum cholesterol levels in high-risk men (Hjermann et al showing a 13% reduction (6); MRFIT showing an 8% reduction (5)), Ahrens concluded that there was little else to recommend the prudent diet over any other diet intervention. "One of the weaknesses of the consensus statement," he wrote, "[was] the failure to acknowledge the paucity of data available to us with regard to risk/benefit ratios of the prudent diet or of the several [dietary] alternatives…"(2).

The current climate of nutrition and public policy is certainly one of "patching up" past errors. Following the panel's recommendation that "the food industry be encouraged to continue and intensify efforts to develop and market foods that will make it easier for individuals to adhere to the recommended diets…" (3), there was a revolution in food production and packaging that led to the development of an abundance of "better-for-you" low-fat and fat-free products, most of which

were higher in refined carbohydrates and some even higher in calories that the original products had been. (Incidentally, Ahrens was the first to provide concrete data showing that high-carbohydrate, low-fat diets caused marked elevations in serum triacylglycerol concentrations, a consequence we are now well-aware of.) Also part of this revolution, saturated fats were replaced by partially hydrogenated oils (*trans* fats) in the majority of manufactured products, an action the food industry is now scrambling to remediate.

Bringing ever-expanding scientific research into the realm of public policy—with its inherent confines and limitations—will never be an easy task. With the complex health and nutrition issues now facing them, policymakers rely increasingly upon scientific advisors to guide their actions. Researchers and scientists currently working in advisory capacities would do well to remember Ahrens' concluding statement—"…As scientists we are expected by the public to render scientifically sound advice. Policy-makers must come to their own conclusions, and will do so for a complex of reasons—political, social, and economic. That is their affair; ours is to be sound, as sound as current evidence permits, stating clearly where the gaps in knowledge exist" (2). ■

1) Ahrens EH Jr. The diet-heart question in 1985: Has it really been settled? *Lancet.* 1985;1(8437):1085-7.

2) Hirsch J. A tribute to Pete Ahrens. *J Lipid Res* 2001;42:891-893.

3) Lowering blood cholesterol to prevent heart disease. National Institutes of Health Consensus Development Conference Statement. *Natl Inst Health Consens Dev Conf Consens Statement*. 1985;5(7):27.

4) Lipid Research Clinics Program. The Lipid Research Clinics Coronary Primary Prevention Trial Results. I. Re-education in incidence of coronary heart disease. *JAMA* 1984;251:351-64.

5) MRFIT Research Group. Multiple risk factor intervention trial. Risk factor changes and mortality results. *JAMA* 1982; 248:1465-77.

6) Hjermann I, Velve Byre K, Holme I. Leren P. Effect of diet and smoking intervention on the incidence of coronary heart disease. *Lanceet* 1981; ii:1303-10.

Refined Carbohydrate Intake Associated with Macular Degeneration

In 2000, an estimated 426,000 cases of legal blindness were attributed to age-related macular degeneration (AMD), a debilitating condition that results in the blurring or complete loss of central vision in one or both eyes. It is estimated that by 2020, the prevalence of AMD will grow to 3 million in the United States alone. The good news is that several nutrients—including zinc, the carotenoids lutein and zeaxanthin, and numerous antioxidants—have shown potential in preventing and/or slowing the progression of this debilitating eye disease. However, new research suggests that dietary factors might also play a role in promoting AMD. Recent cross-sectional data from the Age-Related Eye Disease Study (AREDS) and the Nutrition and Vision Project of the Nurses' Health Study suggest that the intake of refined carbohydrates might be associated with the onset and progression of AMD. Given that most of the energy from carbohydrates in the Western diet comes from foods made from highly-processed and refined grains, understanding the association between refined carbohydrates and AMD is imperative.

To evaluate the influence of refined carbohydrate intake on the risk of AMD, researchers followed 3,977 individuals already enrolled in the ongoing AREDS study. These participants were between the ages of 55 and 80 years at baseline and were followed for an average of 5.4 years. Researchers gathered dietary intake information at baseline using a validated, 90-item food frequency questionnaire (FFQ), which evaluated information about participants' usual dietary intake for the past year. Total daily carbohydrate was estimated from the FFQ and a dietary glycemic index (dGI) score was generated for each individual (weighted average of the GI scores for each food item reported). Although the practical value of the GI is widely debated, it is, by nature, an indicator of carbohydrate quality because it provides a measurement of the influence of an individual food item on postprandial blood glucose levels. In general, refined carbohydrates—prevalent in the typical Western diet—have higher GI scores than do carbohydrates from unrefined sources, thus a higher dGI score would indicate more frequent consumption of refined carbohydrates. Carbohydrate intake and other dietary factors were adjusted for total energy intake.

Potential covariates included age, sex, education level, race, body mass index, smoking status (never or ever), alcohol intake, sunlight exposure, history of hypertension, baseline AMD classification, lens opacity, refractive error, and energy-adjusted dietary variables (total intake of carbohydrate, fat, lutein and zeaxanthin, folic acid, niacin, riboflavin, thiamin, beta-carotene, vitamin C, vitamin E, and zinc). Evidence of early AMD was measured in both eyes for each participant at baseline. Eyes were categorized into one of 5 groups based on type and severity of AMD; group 1 having the least extensive and group 5 having the most extensive evidence of AMD. Only eyes that were categorized within groups 1, 2, and 3 were included in the study because groups 4 and 5 were considered "end-stage" AMD. Progression of AMD within each eye was measured over the course of the study and was defined as progression from one AMD score to the next (ex. 2 to 3). Participants were also categorized by dGI status, which was determined by whether the dGI of each was above (high-dGI) or below (low-dGI) the sex median (77.9 for women and 79.3 for men).

There was no significant difference at baseline between the high- and low-dGI groups with regard to the distribution of age, sex, smoking status, sunlight exposure, lens opacity, or AREDS

treatment. Overall, participants in the high-dGI group were more at-risk for AMD progression than were those in the low-dGI group, and the strength of this effect increased with AMD risk category at baseline. The crude RRs (95% CI) for groups 1-3 were 1.04 (0.92, 1.18), 1.09 (0.93, 1.26), and 1.14 (1.00, 1.29), respectively, with an RR of 1.08 (1.00, 1.17) for the overall sample. The multivariate-adjusted RRs demonstrate that overall, the risk of AMD progression for participants in the high-dGI group was significantly greater than for those in the low-dGI group (RR: 1.10; 95% CI: 1.00, 1.20; P = 0.047).

The adjusted RRs by AMD risk category at baseline showed that eyes categorized with more advanced AMD grades at baseline had greater dGI-associated risk (P for trend <0.001). Groups 1, 2, and 3 had 5%, 8%, and 17% greater dGI-associated risks, respectively. Comparing just the highest and lowest 20% of dGI, risk of AMD progression was observed to be nearly 40% greater for those in the highest 20% (RR: 1.39; 95% CI: 1.08, 1.79; P = 0.012).

This was the first prospective trial to demonstrate a clear association between intake of refined carbohydrates and the risk of AMD progression. It was also observed that those with more advanced AMD at baseline experienced more deleterious effects from high intake of refined carbohydrates. These findings have important public health implications in light of the aging baby boomer population and the growing prevalence of AMD. ■

Chiu CJ, Milton RC, Klein R, et al. Dietary carbohydrate and the progression of age-related macular degeneration: a prospective study from the Age-Related Eye Disease Study. *Am J Clin Nutr* 2007;86:1210-8.

EDITORIAL

Good News, Bad News

It is fortunate for Mr. Gary Taubes that his career does not depend on research funding…If it were, his new book, Good Calories, Bad Calories, would certainly qualify as a "grant killer." In my forty plus years in diet and cholesterol research, I've never seen an individual make the kind of impact he has made by upfront criticism of our national obsession with fat and cholesterol. When I read Taubes' account of the political maneuvering involved in our collective shift to a low-fat, high-carb diet, it was—as Yogi Berra would say—"deja vu all over again." It brought back many memories from those years of scientific, and often not-so-scientific, debates. Taubes does a phenomenal analysis of the scientific and political contributors to the development of the diet-heart disease "consensus" and rightly points out that "the totality of evidence was defined as only those data that confirmed the hypothesis" and that contradictory observations would be ignored for "being inconsistent with the totality of the evidence." Sounds a bit like "I know the truth, don't confuse me with the facts!"

As Taubes notes, according to one of the founders of science, the 16th century Sir Francis Bacon: Good science is rooted in reality, so it grows and develops and the evidence becomes increasingly compelling, whereas "wishful science" flourishes most under its first authors before going downhill. In other words, good science survives the test of time.

Has the rationale for dietary cholesterol and egg restrictions survived the test of time? Not even close! What study has shown a positive relationship between egg intake and heart disease risk? Now, I know you cannot prove a negative so I'm stuck with a bunch of "no significant relationship" studies, but you'd think that at least once in a while, a study would find a relationship if one existed.

In one interview, Mr. Taubes stated that he especially admired the iconoclasm of Dr. Edward H. "Pete" Ahrens Jr., a lipids researcher who spoke out against the McGovern committee's report. Mr. McGovern asked Dr. Ahrens at a hearing to explain his doubts in light of a survey showing that the low-fat recommendations were endorsed by 92 percent of "the world's leading doctors." "Senator McGovern, I recognize the disadvantage of being in the minority," Dr. Ahrens replied. And he pointed out that most of the doctors in the survey were basing their views on indirect knowledge because they didn't actually work in the field of diet and lipid metabolism. "This is a matter," he continued, "of such enormous social, economic and medical importance that it must be evaluated with our eyes completely open. Thus I would hate to see this issue settled by anything that smacks of a Gallup poll." Pete Ahrens was a very wise man who believed that a giant "experiment" was being conducted on the American public and that once that experiment started, it would be very difficult, if not impossible, to correct the direction of our dietary evolution. Taubes, I think, would agree. ■

"This is a matter of such enormous social, economic and medical importance that it must be evaluated with our eyes completely open. Thus I would hate to see this issue settled by anything that smacks of a Gallup poll."

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

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Egg Nutrition Center
1900 L Street NW, Suite 725
Washington, DC 20036