N U T R I T I O N

closeUP

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

Writer/Editor:
Jenny Heap, M.S.

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

The Story on Diet and CHD: Lessons from the Big Picture

For most Americans, it is not at all surprising to hear that in the year 2000, one out of every five deaths in the US was related to coronary heart disease (CHD). With over one million new and recurrent cases of heart attack each year, many Americans live with the specter of CHD looming in their future. They hear about it on the nightly news, read it in the morning paper, hear it *again* in the doctor's office, and are continually buffeted with seemingly endless dietary advice on how to beat the odds. Nearly a century of science and literally decades of dedicated research have brought us to our current understanding of CHD risk. Researchers have poured valuable time and resources into identifying dietary patterns that may help prevent—and those that likely promote—this widespread malady.

So…what do we know so far? This is precisely the question that Walter C. Willett, MD, DrPH and Frank B. Hu, PhD of the Harvard School of Public Health set out to answer in a meta-analysis of research dating back to 1908. Although Willett and Hu acknowledge that there are still no definitive answers, the science appears to be coming closer to a consensus in some areas that were equivocal ten years ago. The following is a summary of their interpretation of the most recent research regarding diet and coronary heart disease:

SATURATED FATS (**SFA**) have long been known to increase total and LDL cholesterol levels. When replacing carbohydrates in the diet, saturated fats also raise HDL cholesterol, however, not enough to favorably modify the LDL/HDL cholesterol ratio. (This ratio appears to be a better predictor of coronary events than LDL levels alone.)

TRANS-FATS, of all dietary lipids, appear to be the most threatening since they raise LDL while lowering HDL cholesterol levels in the blood. Recognizing that this results in nearly double the increase in the ratio of total to HDL cholesterol potentiated by saturated fat, replacing saturated fats with *trans*-fats may actually increase CHD risk. *Trans*-fats have been shown to increase plasma triacylglycerol (TAG) and to impair endothelial function. In addition, research has shown that *trans*-fats impair the critical desaturation processes involved in essential fatty acid metabolism by inhibiting the enzymatic activity of delta-6 desaturase. *Trans*-fats are also thought to contribute to insulin resistance and the development of type 2 diabetes.

POLYUNSATURATED FATS (**PUFA**) tend to decrease total and LDL cholesterol levels. When replacing carbohydrates in the diet, PUFA also increases HDL cholesterol (improving the

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LDL/HDL cholesterol ratio) and decreases TAG, leading to an improved overall lipid profile.

Monounsaturated fats (MUFA), like PUFA, tend to elevate HDL and decrease LDL cholesterol when they replace carbohydrates in the diet, improving the LDL/HDL ratio and reducing triglycerides, thus favorably modifying the lipid profile overall.

Omega-3 fatty acids have been associated with decreased incidence of secondary heart attacks, especially fatal attacks. It is thought that omega-3 fatty acids protect against CHD by decreasing serum TAG, preventing cardiac arrhythmia, conferring anti-thrombotic properties, and improving endothelial function. Omega-3 fatty acids from both fish and plant sources appear protective.

Cholesterol. The Nurses' Health Study, which included multiple dietary assessments for 80,082 women, found that neither dietary cholesterol nor consumption of one egg per day was associated with CHD or stroke after a 14-year follow-up. Similar data were reported from the Health Professionals Follow-up Study, which found no significant association between cholesterol intake and risk for myocardial infarction or fatal CHD.

Folate. Adequate folate intake has been implicated in the prevention of CHD, probably due to its regulation of serum homocysteine.

Carbohydrates, when used to replace SFA in the diet, reduce LDL and HDL cholesterol levels proportionally, resulting in no net change in the LDL/HDL ratio. Simple carbohydrates also elevate serum TAG levels when they replace fats in the diet. Food products billed as low-fat are often higher in simple sugars than their conventional counterparts. Many consumers desiring to limit fat intake have subscribed to this dietary strategy; unfortunately, simply replacing fats with carbohydrates does not appear to be effective in reducing CHD risk.

Fiber. Several epidemiological studies have shown that higher intake of whole grains (vs. refined grains) is associated with lower CHD risk. Aside from its known plasma cholesterol lowering effects, fiber is also recognized for its ability to mediate the insulin response. The process of insulin regulation is thought to influence the development of insulin resistance and type 2 diabetes, which are both associated with the development of CHD. Therefore, fiber may have a double-edged approach to cutting CHD risk.

Hu FB, Willett WC. Optimal diets for prevention of coronary heart disease. JAMA 2002;288(20):2569-2578.

Ascherio A, Rimm EB, Giovannucci EL. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. Br Med J 1996;313:84-90.

Key messages

To reduce the risk of coronary heart disease, Drs. Willett and Hu recommend three dietary modifications:

- Use unsaturated fats (found in vegetable oils, nuts, seeds, avocados, etc...) to replace saturated fats and *trans*-fats in the diet. Saturated fats include butter, lard, and the fat found in beef and other meats. *Trans*-fats are found in foods such as margarine, vegetable shortening, and many prepackaged convenience foods such as crackers and cookies.
- Increase intake of omega-3 fatty acids from fish and/or plant sources. Salmon is one of the best fish sources of omega-3 fatty acids. Flax seed and canola oil are good plant sources.
- Be sure your diet is high in fruits, vegetables, nuts, and whole grains; limit refined grains.

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

Moderate-Protein vs. High-Carbohydrate Diets:

Influence on Body Composition and Lipid Profile in Overweight Women

For decades, high-carbohydrate, low-fat diets have been the weapon of choice in America's battle against heart disease and obesity. Although concerns persist that diets high in animal protein (because they also tend to be higher in saturated fat) may unfavorably affect blood lipids, recent research indicates that moderate-fat diets with a higher protein to carbohydrate ratio may confer some unexpected benefits.

With recent debates over whether high protein diets should have a place among the more conventional therapies for weight loss, the protein vs. carbohydrate rivalry requires head-to-head comparisons. Layman and colleagues tested hypocaloric moderate-protein and high-carbohydrate diets for improving body weight, blood cholesterol levels, and body composition. In a ten-week clinical intervention trial, they studied the effects of diets with high vs. low carbohydrate (CHO)/protein ratios in overweight women.

Twenty-four women between the ages of 45 and 56 were recruited for this ten-week weight loss study. All participants were more than 15% over their ideal body weight. The women were assigned to one of two dietary intervention groups. After collection of baseline data (including a 3-day diet record, body composition, blood lipid profile, glucose, and insulin levels), participants began a four-week controlled dietary regimen, with all meals being provided directly from the research laboratory. Participants followed their respective diet regimens at home for the remaining six weeks, reporting back weekly for remeasurements. Participants were asked to maintain their normal levels of physical activity throughout the study.

Women assigned to the Protein Group (n=12) were provided a moderate-protein diet (1.6 g/kg·day) with a CHO/protein ratio of ~1.4. Women in the CHO Group (n=12) were assigned a high-CHO diet (0.8g/kg·day) with a CHO/protein ratio >3.5. Dietary fat was limited to <30% of calories for both groups. Both diets were designed to fall within the guidelines of the American Heart Association's STEP 1 diet and provided ~1700 kcals total energy, ~50 g total fat, and ~20 g fiber. The protein regimen emphasized the use of animal proteins in place of refined grains and starches and required participants in this group to consume beef in no less than 7 meals per week. The CHO Group was patterned after the USDA Food Guide Pyramid, emphasizing cereal grain products. The Protein Group diet was higher in SFA and contained over twice the amount of cholesterol consumed by the CHO Group. For both groups, dietary SFA intake decreased from baseline.

At the conclusion of the 10-week study, body composition, blood lipid profile, and food records were again measured and evaluated. The macronutrient composition of the Protein and CHO Group diets were 30% protein, 41% carbohydrate, 29% fat, and 16% protein, 58% carbohydrate, and 26% fat, respectively. Both dietary interventions resulted in weight loss. Though the actual weight reduction did not differ between groups, significant differences were observed when the weight loss was categorized by fat and lean mass. Women in the Protein Group lost more body fat (14.4% vs. 12.2%, $P<0.05$) and tended to conserve more lean mass, proportionally, than their counterparts in the CHO Group (lean mass reduction of 0.88 ± 0.33 kg vs. 1.21 ± 0.58 kg, $p=0.07$).

Although blood lipids were significantly higher at baseline for women in the Protein Group, lipid profiles were favorably modified in both groups. After four weeks of the controlled laboratory diet, women in the Protein Group had reduced their total cholesterol by 10.0% and their LDL cholesterol by 10.5%. Likewise, women in the CHO Group had experienced reductions of 11.2% and 14.3% in total and LDL cholesterol levels, respectively. For women in the Protein Group, the HDL/total cholesterol ratio was reduced by 10% compared to an 8% reduction in the CHO Group. In addition, fasting TAG levels declined significantly for women in the Protein Group. No reduction in TAG levels was observed for the CHO Group. HDL cholesterol was increased at week 4 for both cohorts, but did not differ from baseline by week 10. These data indicate that high-protein diets do not unfavorably influence blood lipid levels compared to high-CHO diets.

The results of this study suggest that for patients desiring to lose weight, moderate-protein diets may provide more desirable changes in body composition than high-CHO, lower-protein diets. Because lean body mass is the single most important contributor to resting metabolic rate, loss of adipose tissue and conservation of lean mass during periods of weight loss should encourage maintenance of a healthy body weight.

Layman DK, Boileau RA, Erickson DJ, et al. A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women. J Nutr 2003;133:411-417.

Fish, Mercury, and Coronary Heart Disease Risk

Observations from several epidemiological and clinical intervention studies have led researchers and clinicians to endorse the cardioprotective properties of omega-3 fatty acids from fish. However, recent concerns about mercury levels in fish have prompted some to question whether mercury contamination may render fish more harmful than helpful where CHD is concerned. Because LDL oxidation, a process central to the development of atherosclerosis, is thought to be potentiated by exposure to mercury, many are concerned that mercury contamination may counteract the benefits of omega-3 fatty acids from fish. Two recent studies have addressed this concern.

The first study analyzed data gathered as part of the Health Professionals Follow-up Study (HPFS), which investigated the influence of dietary intakes on CHD risk among US male dentists, veterinarians, pharmacists, optometrists, osteopathic physicians, and podiatrists. The sample population was comprised of 51,529 men between the ages of 40 and 75. Food frequency questionnaires were completed by study participants in 1986 and toenail samples were collected in 1987 to analyze for trace element content. Endpoints included incidents of fatal CHD, nonfatal MI, coronary-artery bypass surgery, and percutaneous transluminal coronary angioplasty between the time of toenail collection and January 31, 1992. Dietary intake was assessed according to a single food frequency questionnaire completed by participants in 1986.

Although toenail content of mercury was significantly associated with estimated fish intake ($r=0.42$, $P<0.001$), no relationship between mercury levels and incidence of CHD could be detected after adjusting for age, smoking, and other traditional risk factors for CHD. Some limitations must be considered, however. The HPFS included only health professionals who could have been engaged in health-promoting activities not taken into consideration in the study such as physical activity and antioxidant intake, which may have attenuated the impact of mercury exposure on CHD risk. The conclusions of this study were also drawn from a single food frequency questionnaire obtained as many as five years prior to the occurrence of any cardiovascular endpoint.

The results from a second multicenter study, reported by Guallar et al., were quite unlike the HPFS findings. Six hundred eighty-four men from eight European countries and Israel were recruited from coronary care units of participating hospitals following a first-time acute MI. Total mercury exposure was determined by analysis of toenail clippings. Adipose tissue samples were obtained from each participant and were analyzed for docohexaenoic acid (DHA) content to estimate long-term fish intake.

Adipose tissue levels of DHA were strongly associated with toenail mercury levels (P for trend <0.001), indicating that fish consumption by this sample population was associated with mercury exposure. Higher mercury levels were also associated with an increased risk for MI (P for trend=0.01). Adjusting for DHA levels, antioxidant levels, and traditional risk factors for CHD resulted in an odds ratio of 2.16 for those with the highest mercury levels (P for trend=0.006). Higher DHA levels were associated with lower risk of MI after adjustment for mercury levels, suggesting that the benefits of fish intake were weakened by the presence of mercury.

Although the results of these two studies appear to be in opposition, some important differences should be taken into account. Since the studies were conducted independently, no comparison could be made between the two groups regarding actual fish intake. Traditionally, European populations consume more fish than their North American counterparts. Thus, the difference observed between the results of these studies might be partially due to higher fish consumption/mercury exposure among the European participants. It is possible that the North Americans in the HPFS did not consume a comparative amount of fish, and thus were not exposed to sufficient mercury to cause a significant increase in CHD risk. Additionally, fish species vary greatly with respect to fat content. Differences between the species of fish consumed in Europe and in North America might have contributed to inconsistencies in study results.

These investigations leave many questions unanswered. Clearly, pregnant women and women of childbearing age who may become pregnant are advised to avoid excessive fish consumption and to eliminate those species of fish found to have the highest levels of mercury (see the FDA's consumer advisory on fish consumption at http://www.cfsan.fda.gov/~dms/admehg.html for more information). However, it is unclear whether fish typically consumed in the US contain sufficient mercury to pose a health risk to other populations desiring to increase their intake of omega-3 fatty acids for heart health.

Guallar E, Sanz-Gallardo MI, van't Veer P, et al. Mercury, fish oils, and the risk of myocardial infarction. N Engl J Med 2002;347:1747-1754.

Yoshizawa K, Rimm E, Morris S, et al. Mercury and the risk of coronary heart disease in men. N Engl J Med 2002;347:1755-1760.

Plasma Homocysteine and Coronary Heart Disease

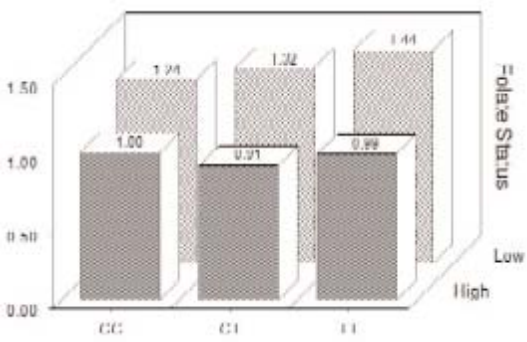
In 1969, McCully noted a high incidence of early vascular disease in children born with homozygous homocysteinuria, a rare metabolic disorder that results in elevated blood homocysteine. However, the possible connection between homocysteine levels and CVD remained unrecognized for decades. Until recent years, cardiovascular research interests have focused on cigarette smoking, blood cholesterol, and blood pressure, which were assumed to be the major predictors of atherogenesis and CVD mortality. However, in the past decade the paradigm has begun to shift toward research of potential alternative predictors of CVD such as homocysteine. Although many investigations have taken place, the results remain contradictory. What can we gain from so much ambivalent data?

In an effort to clear up some of the confusion, the Homocysteine Studies Collaboration was organized to examine 30 prospective and retrospective studies that had investigated the influence of homocysteine levels on ischemic heart disease and stroke. Twelve prospective studies (in which homocysteine levels were assessed prior to onset of CVD incidence) were analyzed separately to eliminate effects of reverse causality. After adjusting for traditional CVD risk factors such as tobacco use, total cholesterol level, and systolic blood pressure, a 25% lower homocysteine level was associated with decreases of ~11% and ~19% in ischemic heart disease risk and stroke risk, respectively. According to the authors, a 25% reduction in plasma homocysteine concentration is the change that would be expected with folic acid supplementation. This modest association between plasma homocysteine and CVD risk, if found to be causal, would support the use of folic acid supplementation to reduce the risk of CHD and stroke.

Many have postulated that elevated homocysteine levels lead to damage of blood vessel walls and thrombus formation; but whether elevated homocysteine plays a causal role in CVD remains to be seen. In the meantime, investigators continue to add evidence to the ever growing case against homocysteine through research related to genetic determinants of homocysteine metabolism.

Methylene tetrahydrofolate reductase enzyme (MTHFR) plays a critical role in the formation of 5-methyltetrahydrofolate (5-MTH), which makes possible the transformation of homocysteine to methionine and keeps serum homocysteine levels in check. The substitution of a cytosine base by thymine (MTHFR 677C to T) in this gene results in a common polymorphism responsible for inhibiting the enzymatic activity of MTHFR. Individuals with this substitution have higher plasma homocysteine and lower folate levels than those without.

CHD Odds Ratios for Various Genotypes by Folate Status



In a meta-analysis of 40 observational studies conducted in Europe, North America, Asia, and Australia, Klerk and colleagues found that individuals with the TT and CT genotypes had higher plasma homocysteine levels and lower folate levels compared to those with the CC genotype ($P<0.05$). Participants who developed CHD also tended to have higher serum homocysteine than those who did not (11.5 vs 10.2 µmol/L, $P<0.05$). Those with the TT genotype were 16% more likely to develop CHD than those with the CC genotype (OR, 1.16; 95% CI, 1.05-1.28).

Interestingly, when folate status was taken into consideration, the TT genotype was associated with increased CHD risk only when folate status was low. Genotype did not appear to make a difference when folate levels were high, suggesting that adequate folate intake might attenuate or negate the elevated risk introduced by the TT polymorphism.

When analysis was undertaken by continent, those with the TT genotype in Europe were more likely to develop CHD than those with the CC genotype; however, this observation did not hold true in North America. This apparent discrepancy might be explained by differences in folate intake between Europeans and North Americans. Although these studies were conducted prior to mandatory fortification of grain products in the United States in 1998, many breakfast cereals in North America had already been routinely fortified for several years. Higher folate intake among North Americans through fortification and supplementation may have adequately compensated for the reduced MTHFR activity in those with the TT genotype.

It is important to recognize that this meta-analysis was conducted using data from several countries, which introduces potential confounding factors. However, the overall results are valuable and warrant further research in this area. The authors suggest that the ease of obtaining adequate folate from the food supply and from supplements might eliminate the need of genetic testing for this polymorphism.

Klerk M, Verhoef P, Clarke R et al. MTHFR 677C T polymorphism and risk of coronary heart disease. JAMA 2002;288:2023-2031.

The Homocysteine Studies Collaboration. Homocysteine and risk of ischemic heart disease and stroke. JAMA

Fish Intake and Stroke Risk

While many investigations have examined the effects of fish intake on ischemic heart disease, relatively few have focused on its close cousin, ischemic stroke. Ischemic stroke, like ischemic heart disease, occurs when a blood clot (thrombus) occludes a vein or breaks free and lodges in a smaller vein (embolus), disrupting the flow of oxygen-rich blood to vital tissues (the brain, in this case). In contrast, hemorrhagic strokes occur when a blood vessel ruptures, causing bleeding in the brain. Long-chain omega-3 PUFA from fish have been shown to inhibit platelet aggregation. While this property is likely one of the most vital in preventing ischemic events, it has been suggested that high fish intake might actually contribute to the risk of hemorrhagic stroke.

Recently, He and colleagues investigated the influence of fish consumption on the risk of ischemic and hemorrhagic stroke in men using data from the Health Professional Follow-up Study (HPFS). Established in 1986, the HPFS is a prospective study of male US health care professionals with 12 years of follow-up. After excluding data for those men who were more likely to experience strokes (those with previously diagnosed stroke, diabetes mellitus, myocardial infarction, or other cardiovascular disease), a total of 43,671 men, aged 40-75 years, comprised this cohort. In 1986, 1990, and 1994, participants completed semiquantitative food frequency questionnaires, including information on fish and other seafood intake. Fish oil supplementation was also assessed for all participants.

The endpoints for this study were episodes of fatal and non-fatal stroke occurring between study initiation and January 31, 1998. All reported strokes were verified by medical records and were classified according to the National Survey of Stroke as ischemic, hemorrhagic, or unknown. Men reporting the highest intake of fish were more likely to have a history of hypertension and hypercholesterolemia, to take aspirin or multivitamin supplements, and to be physically active. They were also more likely to be nonsmokers and less likely to be overweight. Because any of these factors could have influenced outcomes, adjustments were made to compensate for their effects.

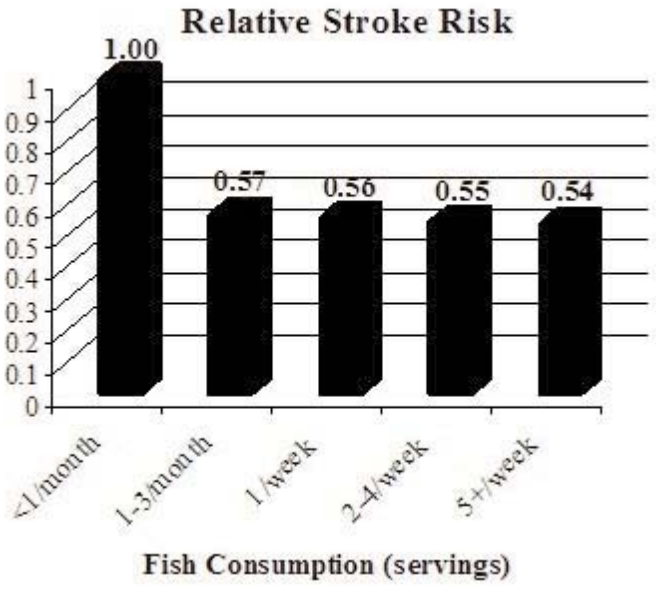
Strokes were reported for 608 of the 43,671 participants. Of these cases, 377 were classified as ischemic and 106 were identified as hemorrhagic, leaving 125 cases unidentified. While the association between fish intake and hemorrhagic stroke was not significant, risk for ischemic stroke was reduced among those reporting fish intake of 1-3 times per month (RR, 0.57; 95% CI, 0.35-0.95) as compared to less than once per month (RR, 1.00). No additional protection was conferred for men who reported consuming fish more than 1-3 times per month.

These data are compelling, however, it is important to avoid making broad recommendations based on one study, especially given the wide confidence intervals associated with the relative risks reported here. Since this investigation was limited to male health care professionals residing in the United States, its findings may not be generalizable to people of every age, race, and gender.

While these data suggest that a few fish meals per month may decrease the risk of ischemic stroke by 40%, it is important to remember that 2-3 fish meals per week are still recommended for the prevention of ischemic events. Since those at risk for ischemic stroke are probably also those at greatest risk for ischemic heart disease, following the latter recommendation is probably the safest bet.

The absence of an association between increased fish consumption and the incidence of hemorrhagic stroke should not be overlooked. This finding is significant as it provides some evidence to support the safety of increasing fish consumption among middle aged American men, who comprise a high-risk population for both ischemic and hemmorhagic events.

He K, Rimm EB, Merchant A, et al. Fish consumption and risk of stroke in men. JAMA 2002;288:3130-3136.



Editorial

It Ain't Over Til the Fat American Loses Weight

Welcome back to NUTRITION WARS! Almost half a century of intense conflict which has yet to determine a winner. The rules are simple: the opponents line up across from each other on the "35% of calories from fat" line and proceed to do battle pushing and pulling the American public between the ultra low-fat goal of one team and the high-protein, high-fat goal of the other. To date, three ten-year periods have been completed and the fourth is just underway. The game will continue until a clear winner is proclaimed by the judges. One quirk of the event is that each period has a new referee who is expected to be open minded and impartial, but such objectivity is rarely displayed. There are four sets of judges: the American Heart Association Nutrition Committee (AHA), National Institutes of Health National Cholesterol Education Program (NIH-NCEP), US Department of Agriculture Center for Nutrition Policy and Promotion (USDA-CNPP), and last but not least, the American consumer (and his/her wallet). The judges use a five points per period scoring system for a total of 20 points per period.

So let's recap the action so far. After three periods you'd still have to call it a toss up. I thought for sure after the third period the fat-devotees were finished and the "sawdust & wood chips" team had them on the ropes. And then all of a sudden, bang, out of the blue, the protein/fat-fanciers found their stride and started a comeback. Maybe the old "rope-a-dope" playing dumb and defenseless started to work! But let's recap each period to see what's really been going on.

In period one (1970-79) it was pretty much a toss up with some strong momentum being shown by each side. Ancel Keys, the father of nutritional epidemiology, was a fair and just referee during that period and called 'em as he saw them. The extremely low-fat, "no-taste-no-matter-what" team was led by Nathan Pritikin, the non-believers followed the guidance of the Food and Nutrition Board "Toward Healthful Diets," and the low-fat, high-carb faithful were whipped into a frenzy by the US Senate McGovern Committee and their national dietary recommendations. By the end of the period the low-fat team had convinced the AHA and they were ahead on points. Score: low-fat 11, low-carb 9.

In the second period (1980-89) the low-fat team was clearly the crowd favorite. They had all the support one could ask for (research grants, committee positions, conventional wisdom, and unwavering consensus, at least from those allowed to discuss consensus). They knocked the living daylights out of the old, tired, staid, conservative fogies who thought proof was needed to back the low-fat, high-carb dietary recommendations. The bodies, and the careers, of the antagonists were strewn across the playing field, and pity any professional who got drafted by the nonbelievers. We saw a new player on the field with Robert Atkins swaying some of the public judges but not the consensus bound scientists. The National Academy of Scientists Institute of Medicine (NAS-IOM) clobbered the opposition with the sheer weight of Diet and Health, and semi-moderates started getting hit from the left with cries for lower fat recommendations. Jeremiah Stamler, whose studies of heart disease risk factors defined the public health approach, tried to referee but his heart was committed to the low-fat diet and the opposition didn't stand a chance. All the judges capitulated and the food industry scrambled to get out as many low-fat, cholesterol-free products as possible. Fat went down, carbohydrates went up and the anti-restriction chorus almost sang its swan song. Score: low-fat 28, low-carb 12.

By the third period (1990-99) it should have been over. AHA, NCEP and CNPP all anointed the low-fat, high-carb team the winner and it only remained for the public to make the vote unanimous. The problem was, the public was having trouble fitting into the judging chairs, and they kept running out of the arena to buy larger clothes. Hard to get a vote when the judges are always out shopping and eating and looking for larger chairs! The captains of the teams kept changing which didn't help matters either. Scott Grundy, a powerful advocate of the diet-heart disease hypothesis, served as the referee for this period but his participation as chair of the HHS-NHLBI-NCEP ATP III committee, participation on the USDA-HHS Dietary Guidelines Advisory Committee, co-chair of the World Health Organization Nutrition Committee, and membership on the Macronutrient Committee of the NAS-IOM caused the high-protein team to get all persnickety about his impartiality. The low, low, low-fat advocates now followed the teachings of Dean Ornish while their antagonists revered Robert Atkins. Somewhere in the mid-low-fat area was the AHA and the teachings of practically every nutrition textbook in the country. It really was all over if they could just get the public to focus and get its mind off 32 oz. soft drinks and super-size meals long enough to get back into the game. Score: low-fat 44, low-carb 16.

With the start of the fourth period (2000-10) it should have been a simple exercise of finishing off a well-thrashed foe. But just when you think its over, it isn't. That's why I hate trying to call a science battle, a great hypothesis ruined by a simple little fact. Religious conflicts are so much easier; beliefs can be manipulated, but facts and knowledge are really unyielding. And when did we start getting referees who question the conventional wisdom? Well someone drafted Walter Willett, another nutritional epidemiologist, to referee the fourth period and all he did was raise issues with what everyone had decided was the truth. And the usual battle

Editorial continued

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between the Ornish-led team and the Atkins-led team continued while the AHA Nutrition Committee decided to shift concepts from prescriptive diets to something called "dietary patterns". Please! The low-fat, high-carb team was way ahead on points and now we start changing paradigms! And where is the American consumer judge (probably out getting *trans*-fatty acid rich French fries)? And what has research to do with a good Nutrition War? High-carb, high-protein, high-fat, metabolic syndrome, obesity, diabetes, physical activity, new risk factors, on and on and on—the complexities just keep getting more complex. The low-fat team had it won and they start to tank in the fourth. Somebody should investigate! There are accusations of bribery by the mega-monolithic, unscrupulous, dastardly food industries to get the low-fat, high-carb team to buckle. Can the low-carb forces make a comeback on points or will it require a knockout? This war can only go on for another decade. After that, we face a sudden death playoff. And the public can't even fit into the stadium seats anymore, let alone planes, buses and trains! And what will we do with all those low-fat (i.e. high sugar) food products when now they want high protein foods? You never know what can happen. But that's what makes NUTRITION WARS must see TV; the uncertainty, the contradictions, the mixing of facts and fiction, and, always important in any competition, the personal opinions and pomposities of those valiant superstars who faithfully do battle day in and day out for their beliefs, their careers, and most importantly, their grants. Stay tuned! Seems pretty clear that we'll have at least one more decade of battles to report and review before a dietary dogma is given the "This Can Assure You Immortal" silver plate award.

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

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

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