"The important thing in science is not so much to obtain new facts as to discover new ways of thinking about them."

—Sir William Bragg

INSIDE

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NUTRITION CLOSE-UP

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"Low-Fat" Diet Advice for Women: Is the Verdict Still Out?

The decades prior to the 1990s were characterized by an intense fear of dietary fat. The theory that dietary fat intake would invariably increase risk of cancer, heart disease, and numerous other maladies was widely accepted. Moreover, the fear that fat would simply make you...well...*fat*...was enough to drive dietary advice focused on avoiding the 9-calorie-per-gram macronutrient at all costs. Observations from epidemiological migration studies supported the assumption that dietary fat was causally related to various diseases of the Western world. Women who had migrated from countries where dietary fat intake (and the incidence of cancer and heart disease) was traditionally somewhat lower than in the US were now beginning to reflect the higher disease rates of their American counterparts. Given the climate of the times, it is surprising to note that it wasn't until the early 90s that a study was designed to evaluate the long-term results of promoting low-fat dietary patterns in a population of free-living mature women.

Organized in 1991-92, the Women's Health Initiative (WHI) was the first randomized trial designed to directly assess the impact of a low-fat dietary pattern on the health of racially and socioeconomically diverse postmenopausal women. In particular, the WHI trial assessed the risks of breast cancer, colorectal cancer, and cardiovascular disease in this population.

We must recognize the difference between simply reducing dietary fat intake and actually following a "low-fat" diet. In this study, the difference was several percentage points...

A cohort of 48,835 women aged 50-79 years, participated in this study. The intervention group consisted of 19,541 women, with a comparison group of 29,294. Women with a history of colorectal, breast, or other cancers within 10 years of study initiation were excluded from the trial, along with those with known type 1 diabetes, terminal illness (with expected death within 3 years), or baseline fat intake of <31% of calories. Because the trial was designed to determine the effects of a low-fat diet in a free-living population, no meals were provided. Instead, women in the intervention group received dietary counseling to follow a low-fat eating pattern defined as follows:

- *<20% of total calories from fat*
- *5 servings of fruits and vegetables per day*
- *6 servings of grains per day*

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For women in the intervention group, dietary fat intake goals (in grams/day) were provided to each woman based on height. Dietary counseling was also provided to help each woman increase her servings of fruits, vegetables, and grain and to adhere to a low-fat dietary pattern. No total calorie intake or weight loss goals were set. Participants in the comparison group received the US Department of Health and Human Services' publication, *Nutrition and Your Health: Dietary Guidelines for Americans*, but were not provided with counseling of any kind, nor were they asked to make any dietary changes.

All participants completed a WHI food frequency questionnaire (FFQ) at baseline and at one year. A third of the women filled out FFQs each subsequent year so that an FFQ was obtained for each participant once in every 3-year period. The women underwent fasting blood draws at baseline, one year, and in 5.8% of the women (2816) at years 3 and 6 of follow-up. The samples were analyzed for plasma total cholesterol, plasma triglycerides, serum γ-tocopherol, and serum total carotenoids at baseline and at the 3-year follow-up.

The women were expected to complete a mammography screening at baseline and again every 2 years for the duration of the study. Electrocardiograms were obtained every three years and women completed a medical update questionnaire every 6 months. (Although bowel exams were not required over the course of follow-up, the frequency of these exams was monitored by WHI administrators.) Medical records were reviewed for each self-reported diagnosis of colorectal cancer, breast cancer, CHD, or stroke, and diagnoses were confirmed by physicians blinded to group assignment.

After one year, the intervention group had decreased their average total fat intake from 37.8 grams to 24.3 grams, an intake 10.7% lower than the comparison group. Only 31.4% of the women had met the goal of 20% of energy from fat at year one of follow-up, and only 14.4% had maintained it by year 6. The women in the intervention did reduce their saturated fat intake to less than 10% of total calories and increased their fruit, vegetable, and grain servings by year 1. They also achieved a significant increase in folate intake and in plasma total carotenoids. Total red meat and vitamin E intake decreased, as did serum total cholesterol and plasma γ-tocopherol levels. Although this was not a focus of the WHI trial, women in the intervention group did not meet the intake levels currently recommended for fiber, fish, or polyunsaturated fat.

The frequency of mammography screening was similar between the intervention and comparison groups. Invasive breast cancer was reported by 655 women (3.35%; 0.42% annualized incidence rate) in the intervention group and 1072 (3.66%; 0.45% annualized incidence rate) in the comparison group (hazard ratio [HR], 0.91; 95% CI, 0.83-1.01 between the two groups). Although the difference in breast cancer risk over the average 8.1 year follow-up period was not statistically significant, the non-significant trends toward risk reduction suggest that further research is warranted in this area. The intervention did not reduce the risk of invasive colorectal cancers (HR, 1.08; 95% CI, 0.90-1.29). In the comparison group, 279 of the women (0.12% per year) vs. 201 women in the intervention group (0.13% per year) reported confirmed cases of invasive colorectal cancers, which is similar to national data for women within this age group (0.12%).

The primary objectives of the WHI were to observe the effects of a low-fat diet on the incidence of breast and colorectal cancer in a large population of ethnically diverse postmenopausal women. As a secondary objective, the WHI also looked at the effects of a reduced-fat diet on the risk of cardiovascular disease. The endpoints were major CHD (defined as acute myocardial infarction [MI], silent MI, or death from CHD) and/or cardiovascular disease (CVD; defined as CHD events plus ischemic and/or hemorrhagic stroke).

By year 3 of follow-up, women in the intervention group had experienced reductions in body weight, BMI, and waist circumference measurements by 1.29 kg, 0.49 kg/m^2, and 0.98 cm, respectively ($P<0.001$). They also experienced reductions in LDL cholesterol levels by 3.55 mg/dL, diastolic blood pressure by 0.31 mm Hg, and factor VIIc levels by 4.29%, on average. Although these changes were statistically significant, they were clinically minor and there were no differences between control and intervention groups with regard to rates of CHD (HR, 0.97; 95% confidence interval [CI], 0.90-1.06), stroke (HR, 1.02; 95% CI, 0.90-1.15), or CVD (HR 0.98; 95% CI, 0.92-1.05). Triacylglycerol (TAG) concentrations, HDL cholesterol levels, total cholesterol:HDL cholesterol levels, glucose and insulin levels, and measures of insulin resistance remained unchanged over the course of follow-up. The researchers did note a trend toward a reduction of CHD risk in women who ate less saturated and *trans*-fat.

In summary, although some risk factors for CVD were modestly improved in this population of postmenopausal women, the reduced-fat intervention did not reduce the risk for CHD, stroke, breast cancer, or colorectal cancers over an average of 8.1 years of follow-up. We must recognize the difference between simply reducing dietary fat intake and actually following a "low-fat" diet. In this study, the difference was several percentage points. While dietary advice to follow a low-fat eating pattern helped these women reduce total fat intake, the reductions were not substantial enough to be considered "low-fat." What was meant to be a "low-fat" diet characterized by intake of <20% of calories from fat in this study evolved into a moderate-fat (~26.7% of calories from fat) diet by year 3. By year 6, the mean total fat intake was up to 28.8% of calories for women in the intervention group and the

comparison group maintained an average intake of 37.0% of total calories from fat (unchanged from baseline). Whether the trends toward risk reduction would have reached significance if the intervention had been initiated in younger women or if the group had further reduced fat intake is not clear. What *is* clear is that conclusions regarding the effects of low dietary fat intake on health risks in women cannot be drawn from these results. ■

Prentice RL, Caan B, Chlebowski RT, et al. Low-fat dietary pattern and risk of invasive breast cancer: The women's health initiative randomized controlled dietary modification trial. *JAMA* 2006;295:629-642.

Beresford SAA, Johnson KC, Ritenbaugh C, et al. Low-fat dietary pattern and risk of colorectal cancer: The women's health initiative randomized controlled dietary modification trial. *JAMA* 2006;295:643-654.

Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: The women's heart health initiative randomized controlled dietary modification trial. *JAMA* 2006;295:655-666.

KEY MESSAGES

In this study, women counseled to follow a low-fat dietary pattern and followed for a period of eight years experienced no reduction in risk of breast cancer, colorectal cancer, or CVD. Three possible explanations for these results are as follows:

- The postmenopausal years might be too late for this (or any) kind of dietary modification to significantly reduce risk of breast cancer, colorectal cancer, or CVD.
- The difference in fat intake was not great enough between groups to yield a statistically significant reduction in risk in the intervention group.
- Lastly, this trial did not differentiate between types of fat eaten. Women in the intervention group were not counseled to replace saturated fat with mono- and polyunsaturated oils, which are now thought to have many health benefits.

Protein, Ghrelin, and Perceived Satiety

The obesity epidemic in the United States and beyond has drawn a great deal of attention among academics, researchers, and policy makers alike. Because a majority of the population stands to benefit from scientific advances in the area of weight control and maintenance, interest in the mechanisms of appetite and satiety has crossed into the realm of popular science. Terms like "leptin" and "cortisol" are becoming more commonplace in communications meant for consumer audiences. Ghrelin, too, is getting its fair share of stage time. This peculiar-sounding hormone was named for a Hindu word meaning, "growth"—and appropriately so. A powerful hormone that stimulates appetite and food intake, it turns out that ghrelin is not so oddly named, after all. Ghrelin plays opposite insulin, leptin, and the gut hormone, PYY, all of which suppress hunger.

Ghrelin production decreases in response to oral intake of glucose and fat and in response to intravenous glucose administration. Meals high in carbohydrates reduce ghrelin levels more effectively than do high-fat meals. Although protein is thought to be the most satiating macronutrient, little is known about its effects on this critical regulator of hunger and food intake.

Blom and colleagues hypothesized that dietary protein enhances satiety by decreasing circulating levels of ghrelin. To test their theory, the research team recruited 15 healthy young men, 18-26 years of age, with BMI values between 19.0 and 25.0 kg/m^2 to participate in a single-blinded dietary crossover trial (randomized for order). The study utilized two dairy-protein based test meals, equal in calories, but differing in protein and carbohydrate composition.

The high-carbohydrate meal (HC) consisted of plain yogurt mixed with 20 g saccharose and 1.5 g acetaminophen (for subsequent estimation of the gastric emptying rate). This meal was high in carbohydrate (47.3% of energy) and moderate in protein (19.3% of energy). The high-protein meal (HP) consisted of a whey-protein enriched dairy product to which 1.5 g acetaminophen and the sweeteners, aspartame and Acesulfame K, were added. This meal was high in protein (58.1% of energy) and low in carbohydrate (14.1% of energy). To isolate the effects of protein and carbohydrate, the test meals were equal in weight, volume, fat and energy composition, viscosity, and taste.

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Each participant was randomly assigned to one of the two test meals, which was consumed for breakfast on the test day. The participants were asked to consume the same foods and beverages the night before both test meals and to record their intake for these evenings. The following morning, participants completed a well-being questionnaire and were weighed prior to eating the test breakfast (HC or HP). Participants finished their test meals in less than 10 minutes and did not eat or drink anything else for a period of three hours. Blood was collected from each participant at 15, 30, 45, 60, 90, 120, and 180 minutes post-breakfast. At each blood-draw, participants completed Visual Analogue Scales (VAS) to gauge their hunger, fullness, and desire to eat. Following the final blood-draw, participants were offered a buffet-style lunch and were instructed to eat until they reached satiety.

Plasma acetaminophen concentrations were used to estimate rates of gastric emptying. Following the HC breakfast, plasma acetaminophen concentrations rose rapidly, indicating that stomach emptying was more rapid following this meal. Following the HP meal, these concentrations rose more slowly (response was ~18% smaller; $P<0.0001$), indicating that the HP breakfast slowed gastric emptying.

Total blood ghrelin concentrations decreased, as expected, following both breakfasts, however, the drop was greater following the HP meal (-18% following HC vs. -25% following HP; $P<0.0001$ for ghrelin response following treatment). The lowest ghrelin measurement was reached at 60 minutes following the HC meal and at 120 minutes following the HP meal.

Glucose concentrations were dramatically different following the HC and HP meals. While serum glucose levels rose ~24% after the HC meal and reached peak values at 30 minutes, they did not rise following the HP meal. Rather, they dropped ~10%, reaching the lowest values at 60 minutes ($P<0.0001$). Insulin concentrations increased following both test meals (~8-fold following the HC meal and ~5.5-fold following the HP meal; $P<0.0001$). These concentrations were lower at 30 and 45 minutes post-breakfast for participants who had eaten the HP test meal. Glucagon concentrations increased ~31% following the HC treatment and ~130% following the HP treatment ($P<0.0001$).

Subjective measures of hunger, desire to eat, and prospective food consumption decreased and measures of fullness increased following both test meals, with no significant difference between treatments. Aside from a reduction in fat intake following the HP breakfast ($P=0.05$), the test meals did not result in significant differences in macronutrient or energy intake during the buffet-style lunch. None of the subjective measures of satiety were associated with ghrelin concentrations. Insulin concentrations correlated with fullness ratings ($r=0.45$; 95% CI: 0.00, 0.74). The AUC (area under the plasma concentration curve) for the ghrelin response did not correlate significantly with fat intake during the lunch following either breakfast treatment.

Aside from the reduction in ghrelin concentrations following the HP breakfast (which was greater than the reduction seen following the HC breakfast), the results were not what the research team had expected. The HP breakfast did not increase subjective measures of satiety or reduce subsequent food intake as projected. Blom and colleagues conclude, however, that with a small cohort of 15 men and an experimental design that lasted just two days, many variables could have come into play to confound these findings. They specifically note that there might not have been sufficient statistical power to detect differences between treatments. The fact that the protein-rich breakfast reduced circulating ghrelin levels to an extent not observed following the high-carbohydrate breakfast suggests that the protein in the meal should increase satiety. Subjective measures of hunger and satiety were obtained during infusions and blood samplings that could have affected the participants' perceptions of these measures. What this study does contribute, however, is a possible mechanism to explain why higher protein intake has been shown, in previous studies, to reduce subsequent energy intake. ■

Blom W, Lluch A, Stafleu A, et al. Effect of a high-protein breakfast on the postprandial ghrelin response. *Am J Clin Nutr* 2006;83:211-20.

READ IT AGAIN... FOR THE FIRST TIME

1981

Dietary Cholesterol and LDL Metabolism

Dietary saturated fat has long been linked to negative cardiovascular outcomes, due mostly to its association with high serum cholesterol. Dietary cholesterol was likewise believed to be related to elevated heart disease risk, due mostly to its association with saturated fat. Since in nature, one rarely occurs without the other, it was easy to assign guilt by association. However, recognizing that reliance upon this assumption would not satisfy the demands of sound science, researchers began to question this hypothesis using cholesterol feeding studies to define the independent effects of dietary cholesterol. But large variations in individual responses to dietary cholesterol made it difficult to arrive at any conclusions regarding the effect of high cholesterol intake on CHD risk. The hypothesis was that dietary cholesterol would increase LDL cholesterol levels by reducing liver LDL receptors, thus decreasing the rate of LDL catabolism, and/or increasing the production of VLDL particles. Ginsberg et al. tested this assumption by assessing VLDL and LDL turnover in healthy, normolipidemic men fed low- and high-cholesterol diets.

The study cohort consisted of five healthy men between the ages of 32 and 35 who were near their ideal body weights and free of any disorders affecting lipoprotein metabolism. Lipid profiles for all participants were within normal limits and none were taking any medications. The study protocol consisted of a baseline phase of 4 weeks duration in which the men consumed a diet of 45% carbohydrate, 40% fat, 15% protein, and 150 mg cholesterol/1000 kcals. This first phase was followed by a washout period of 3-8 months, after which the intervention phase began. In this phase, the men consumed a diet identical to that consumed in the baseline phase except the amount of cholesterol consumed increased to 500 mg/1000 kcals (given in the form of egg yolk). All meals during the baseline and intervention phases were prepared in the Clinical Research Center (CRC) and calories were adjusted as necessary to promote weight maintenance.

Blood was drawn from each participant after the first week of the baseline phase to isolate LDL and VLDL particles for radio-labeling used in the turnover studies. Fasting blood samples were obtained from participants each day for the final two weeks of each diet phase to measure total, LDL, and HDL cholesterol levels as well as total triacylglycerol (TAG) concentrations. These measures were determined by averaging 3 to 5 samples taken during this period.

None of the participants experienced statistically significant changes in serum total or LDL cholesterol levels as a result of the greater than 3-fold increase in dietary cholesterol during the intervention phase. All were categorized as "nonresponders." Plasma TAG and HDL cholesterol concentrations likewise remained stable over the course of the intervention, as did the ratio of cholesterol to TAG in VLDL. Finally, the mean catabolic and production rates of VLDL apoB and LDL apo B also remained similar over the course of both diet phases.

Overall, the mean plasma concentration and turnover rate of VLDL TAG remained stable over the intervention phase. One participant experienced an increase in the rate of VLDL TAG production and catabolism, but his plasma concentration did not change, indicating that the catabolic and production rates increased to a similar extent and remained in balance.

Although many studies have demonstrated an association between saturated fat and plasma cholesterol levels, this study supports several previous cholesterol feeding trials in which additional dietary cholesterol (independent of dietary saturated fat) was not associated with changes in plasma cholesterol concentrations. Ginsberg and colleagues concluded that an increase in biliary cholesterol secretion and/or a downregulation of endogenous cholesterol synthesis play important roles in maintaining stable plasma cholesterol levels in "nonresponders" to increased dietary cholesterol. Subsequent studies have shown that the major regulatory response is a decrease of endogenous cholesterol synthesis. ■

Ginsberg H, Le N, Mays C, Gibson J, Brown WV. Lipoprotein metabolism in nonresponders to increased dietary cholesterol. *Arteriosclerosis* 1981;1:463-470.

High Cholesterol Intake Influences Chylomicron Remnant Metabolism

Extensive research on the effects of saturated fat intake on serum lipid metabolism has shown a clear association between saturated fat intake and blood cholesterol levels. With a few exceptions, most foods that contain saturated fat are also high in cholesterol. The relative influences of dietary saturated fat and cholesterol are therefore frequently assumed to be similar. Few studies have documented the independent effects of dietary cholesterol on markers of cardiovascular disease (CVD) risk. A number of serum lipoproteins besides total cholesterol and LDL cholesterol have been implicated in CVD risk, including chylomicrons. These lipoproteins are synthesized in the intestine to transport absorbed fat and cholesterol in the bloodstream. It is thought that slower rates of chylomicron catabolism promote atherosclerosis. Although the accumulation of chylomicron remnants (CR) in the serum has been associated with coronary artery disease, the effects of dietary cholesterol—independent of saturated fat intake—on chylomicron remnant clearance rates have not been examined.

Egg yolks are high in cholesterol, yet contain very little saturated fat. To determine the independent effects of cholesterol intake on chylomicron metabolism, César et al. examined the effects of high egg intake on CR clearance in 25 young male volunteers. Participants were between the ages of 17 and 22 and had serum total cholesterol levels between 140 and 210 mg/dL. Those who smoked or drank alcohol heavily were excluded from the study, as well as sedentary men and those using prescription drugs.

Participants were expected to adhere to the NCEP Step I diet during both treatment periods. Those in the EGG group were asked to consume three whole eggs (providing a total of 640 mg cholesterol) each day for the duration of the treatment period. Participants in the SUB group consumed the equivalent volume of a fat-free, cholesterol-free egg substitute daily (providing 0 mg cholesterol per day).

Each participant was randomly assigned to adhere to a low- or high-cholesterol diet (LCD or HCD) for a period of 15 days. Both diets were composed of 60% carbohydrate, 15% protein, and 25% total fat (7% saturated fat + 18% monounsaturated and polyunsaturated fatty acids). Three egg whites/day were added to this base diet for the LCD treatment and three whole eggs/day were added for the HCD treatment. This trial was not designed as a cross-over study, so participants followed only one diet.

Dietary history and physical activity were assessed pre-study to determine the calorie needs of each participant and meals were planned accordingly. Meals were prepared and distributed through the school cafeteria for all days of the study (including weekends). The study administrators used a 15-day weighed food record to determine actual intake of calories and macronutrients. Leftovers were weighed and subtracted. Compliance with the dietary treatments was not reported in this article. CR clearance rates were determined by compartmental analysis and blood lipoprotein concentrations were measured on the day after study completion.

The two study groups were similar at baseline with regard to serum lipid and lipoprotein profiles and physical characteristics. Actual intake as assessed by the study administrators did not differ between diets with regard to dietary macronutrient or energy intake. Both were low in total and saturated fat. The only difference between diets was the total cholesterol intake ($P<0.05$).

For the HCD group, total and LDL cholesterol, as well as Apo B concentrations, rose in comparison to the LCD group ($P<0.05$). HDL cholesterol also rose significantly for participants in this group, so the LDL/HDL ratio did not differ between treatments. Triacylglycerol, Apo AI, and Lp(a) concentrations remained similar between treatment groups. According to data from compartmental analysis, the fractional clearance rate of the C-CE emulsion was 52% lower in the HCD than the LCD group ($P<0.001$).

This study indicates that in a cohort of young Brazilian men with normal blood lipid profiles, high daily cholesterol intake (average of 804±40 mg/day), independent of saturated fat intake, prolongs the residence time of chylomicron remnants in the plasma, a factor currently thought to promote atherogenesis. Impaired chylomicron remnant clearance rates have been associated with obesity, diabetes, and other characteristics of the metabolic syndrome, along with other risk factors for atherosclerosis, many of which are associated with abnormalities of dietary and endogenous triacylglcerol metabolism. The high cholesterol intake also raised LDL and HDL cholesterol levels such that the LDL/HDL ratio was the same between the high- and low-cholesterol groups. This indicates that at least for this parameter, the higher cholesterol intake did not increase CAD risk. Further research in this area is warranted to clarify the role of chylomicron remnant clearance rates in the atherogenic process. It is also unclear whether chylomicron remnants enriched with cholesterol, which would be cleared by the liver, have the same characteristics as remnants varying in fat saturation, which would be metabolized primarily by adipose and muscle tissues. A randomized cross-over design with a larger number of participants would be useful in clarifying the effects of cholesterol intake on chylomicron remnant clearance. ■

César TB, Oliveira MRM, Mesquita CH, and Maranhão RC. High cholesterol intake modifies chylomicron metabolism in normolipidemic young men. *J Nutr* 2006;136:971-976.

EDITORIAL

A Burnt Out Bulb in the Guiding Light

Feeling a little lost lately? An unnerving sense of disorientation? Maybe a bit dazed and confused? Don't feel alone, everyone in nutrition is feeling it! What is it? A deep and abiding sense of doom and gloom because they've been upsetting the apple cart! Someone shook the foundations of our long held beliefs! Someone intentionally violated the prime directive! Oh the uncertainty, the ambiguity, the mental anguish. How could science so easily violate our hard fought-for and long-established beliefs?

The prime directive in nutrition is "don't ask, don't tell." Don't ask questions about the established nutrition dogmas and don't tell anyone that you harbor doubts of their validity. In the past, if you violated this prime directive, the Lord High Nutrition Inquisitioner would bar you from committees, ban you from funding, and make sure that your heretical thoughts would not damage the nutrition community. You would be ostracized from the higher powers to avoid intellectual contamination of thought. You were out! Marginalized! Untouchable! And you were often accused of the ultimate sin—accepting industry funding!

Not so today. Our contentious red state, blue state mentality has permeated down to even the scientific community and those who may have been silenced in the past are joining forces to assure that their messages are heard, and heard by those who count. No longer will research papers suggesting that not all is well in the world of dietary guidance be shuttled aside to the bins of "unobserved publications" but rather appear in front line journals with immediate (and too often inaccurate) media attention. No longer will questioning the conventional wisdom be viewed as an act of treason to the King of Pyramids or rainbows or plates or whatever.

Remember the first epidemiological study showing no relationship between dietary fat and breast cancer. The anger, the angst, the accusations! Bad science! Hidden agendas! Yada, yada, yada! Remember the convulsions over data showing that maybe, just maybe, there might be something to the low-carb, high-protein diet. Worse yet, plasma cholesterol levels didn't skyrocket to instant myocardial infarction levels on those fat-laden deadly diets. No way, Jose. Something's obviously out of alignment and the gods of nutrition are getting malevolent.

All fats are bad; well, some fats are okay but others are really bad. Low-fat diets will lead to weight loss (so much for that idea). Dietary cholesterol increases heart disease risk, we just haven't been able to prove it yet (precautionary principal at its best). Diabetics shouldn't have sugar (at least that one got changed). One diet fits all (ever hear of nutrigenomics?). The Women's Health Initiative will show the health benefits of a low-fat diet (not impressed yet). Carotenoids and cancer; vitamin E and heart disease; folate and homocysteine levels; etc, etc, etc. Why are so many of my fundamental beliefs unprovable and being challenged?

The tragedy of science: a beautiful hypothesis demolished by an ugly fact. Today we seem to be getting more of the ugly facts than we can handle. So many changes in our thinking. So many shifts in conventional wisdom. It's much easier to stick with the old nutrition commandments than change to (dare I suggest it) treating the individual patient. It has been said that change = risk + opportunity. The risk is that if we are unable to accept the new, we stay with the old, and the patient has ineffective therapy. The opportunity is that we might actually treat the risk factors with effective interventions that do some good. Guidelines must change with the times, with the evidence, and with the recognition that nutrition is a dynamic science—not mired in the opinions of those from forty years ago who had limited evidence to work with. And don't give me the argument that we'll only confuse the public; they're already confused and steadily losing their belief in the validity of our chronic harping on saturated fat, *trans*-fat and cholesterol.

I wonder, given the evidence we have today, as compared to forty years ago, if an extensive analysis of the data in its totality (not just the past five years) would result in the same set of dietary recommendations we have lived with for forty years. Wouldn't it be fun to find out! ■

> "A scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die and a new generation grows up that is familiar with it."
>
> —Max Planck

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

"The incredible, edible egg..."

Sound familiar? Well, it should...

This catchy jingle has served as the slogan of the American Egg Board for the past 30 years. And this year, as the American Egg Board celebrates its 30th anniversary, its slogan has been nominated to the Madison Avenue Advertising Walk of Fame, sponsored by Advertising Week, the largest gathering of advertising industry professionals in North America.

Please join us in celebrating 30 wonderful years at America's breakfast table by visiting http://advertising.yahoo.com/advertisingweek_06.com and voting for the "incredible, edible egg" as America's favorite slogan.

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