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I N S I D E

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Low-Carb Diets for Overweight Adolescents: Big News or Bad Idea?

Recent years have seen a remarkable surge of scientific interest in the use of high-protein, low-carbohydrate diets in weight-loss programs. Researchers have focused on the effects of these diets on both weight loss and changes in blood lipids in adults. The majority of these studies have found that low-carbohydrate, low-calorie diets—as compared with traditional low-fat, low-calorie diets—result in greater weight loss without negatively impacting the lipid profile. With the already high and increasing rate of obesity among US children and adolescents, and the premature onset of diseases related to obesity, interest in using low-carbohydrate diets in overweight pediatric populations has emerged. Researchers at Schneider Children's Hospital in New York set out to address the question of whether overweight adolescents could benefit from this kind of dietary intervention.

Participants were recruited from the young patient population referred for weight concerns by their pediatricians to the Center for Atherosclerosis Prevention of Schneider Children's Hospital. The group was comprised of 39 overweight adolescents (BMI >95th percentile for age), ages 12-18. This group was divided into two study cohorts. During the first two weeks of the study, the low-carbohydrate group (LC; n=20) was instructed to follow a diet consisting of ≤ 20 g CHO/day. Protein, fat, and energy were to be consumed as desired. Participants in the LC group were also asked to consume at least 50 oz of water per day and to take an age-appropriate multivitamin supplement. From weeks 3 to 12, the carbohydrate limit was raised to 40 g CHO/day, to allow for additional nuts, fruits, and whole grains. For the duration of the study, the low-fat, conventional diet group (n=19) was instructed to consume <40 g fat per day, 5 servings of starch (15 g CHO/serving or a total of 300 kcals from starch; whole grains encouraged, fruit juices excluded), and fruits, vegetables, and fat-free dairy products at will. An age-appropriate multivitamin supplement was also recommended for participants in the LF group. Participants from both groups were encouraged to engage in 30 minutes of aerobic exercise three times a week.

Height, weight, and BMI were measured at baseline and monitored twice a week for the duration of the study. Serum lipids for all participants were measured at baseline and after 12 weeks to detect changes in fasting total cholesterol (TC), triacylglycerol (TAG), HDL cholesterol (HDL), non-HDL cholesterol, and calculated LDL

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cholesterol (LDL). Glucose, urea nitrogen, creatinine, urea nitrogen/creatinine ratio, total protein, albumin, total bilirubin, alkaline phosphatase, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and electrolyte levels were also measured at the beginning and end of the study. Both groups were asked to keep a daily record of urinary ketones to monitor macronutrient metabolism. Total calories, fat, carbohydrate, and protein were monitored by a registered dietitian to assess individual dietary compliance. Intake of cholesterol and saturated, monounsaturated, and polyunsaturated fatty acids were also measured. Participants did not differ significantly on any measure before initiation of the intervention.

Despite the fact that the LC group, on average, consumed more energy and fat than the LF group, the LC group was more successful in reducing body weight. The average reduction in BMI for LC participants was 3.3 ± 3.0 kg/m^2, significantly greater than the 1.5 ± 1.7 kg/m^2 average for LF participants. Average weight loss for the LC participants was 9.9 ± 9.3 kg as compared to the 4.1 ± 4.9 kg weight reduction for LF participants. All 16 LC participants who completed at least 8 weeks of the study lost weight. All but one participant in the LF group also lost at least some weight. However, 8 of 16 LC participants lost >1 kg/wk, compared to 4 of 14 participants in the LF group.

Results of previous studies suggest that low-carbohydrate diets result in a reduction of adipose tissue and maintenance of lean mass. An important limitation to this study is that it did not monitor changes in body composition, thus, no conclusion could be made regarding the source of weight loss for these participants. Whether the weight lost was composed mainly of adipose tissue, lean tissue, or fluid could not be determined.

A major concern with promoting a low-carbohydrate diet with unrestricted energy intake from fat and protein is the potential impact on serum lipids. Adolescents in the LC group reported consuming significantly more saturated fat and cholesterol than their LF counterparts. Despite this, no component of the lipid profile worsened significantly for this group. While serum LDL cholesterol concentrations dropped for adolescents in the LF group, they did not change significantly for those in the LC group. However, while serum TAG (an independent predictor of CHD risk) remained constant for the LF group, it declined significantly for LC adolescents (-48.3 ± 29.0 mg/dL). Non-HDL cholesterol decreased significantly for both groups, with the LC group experiencing a much steeper decline than the LF group. The LC diet regimen was associated with increases in serum HDL cholesterol.

Although the degree of ketosis was not monitored, adolescents in the LC group reported urinary ketones (a marker of the breakdown of fat for energy) almost daily for the duration of the study. None of the adolescents on the LF diet reported urinary ketones at any time during the study. The main side effects reported by LC participants were constipation or diarrhea (3 of 16) and headache (2 of 16). Two of the 14 LF participants reported fatigue. Participant drop-out rates were similar for both groups (4 of 20 in the LC group and 5 of 19 in the LF group). Discomfort with the idea of consuming a majority of calories from fat was the major reason for LC participants dropping out of the study (2 of 4). For LF participants, limited food choices (2 of 5) and noncompliance (2 of 5) were the most common reasons given for discontinuing participation.

The authors conclude that for overweight adolescents with normal lipid profiles (or for those whose primary concern is hypertriglyceridemia or low HDL cholesterol), a low-carbohydrate diet such as the one used in this study could be beneficial. They cautioned that perhaps those adolescents with elevated LDL cholesterol levels would benefit more from the conventional low-fat diet recommended by the National Cholesterol Education Program (NCEP). This study provides valuable information about the short-term effectiveness of non-calorie-restricted, low-carbohydrate diets in aiding weight loss in the overweight adolescent population.

Sondike SB, Copperman N, Jacobson M, et al. Effects of a low-carbohydrate diet on weight loss and cardiovascular risk factors in overweight adolescents. *J Pediatr* 2003;142:253-8.

Key messages

- Short-term, low-CHO diets with no fat or energy restrictions appear effective in promoting weight loss in overweight adolescents.
- The low-CHO diet did not have a negative impact on serum lipids of overweight adolescents. Although LDL concentrations remained constant, TAG levels decreased and HDL levels increased for this group.
- Drop-out rates were similar for the low-CHO and low-fat diet groups.

Obesity Genes Sensitive to High-CHO Diets?

The advent of genetic research has resulted in a heightened awareness of the need for novel approaches to health maintenance and disease prevention for individuals. New research consistently repeats the message that genes and environment—including the food environment—are mutually influential and can significantly impact phenotypic expression. Among scientists, there is an emerging recognition that the one-diet-fits-all approach has some inherent limitations in our genetically diverse population. For example, researchers have reason to think that the well-intentioned efforts of some dieters following low-fat, high-carbohydrate diets might be shortchanged by their own genes. A polymorphism (known as Gln27Glu) affecting the ß2-Adrenergic receptor gene (ß2-AR), known to be involved in lypolysis and adipocyte lipid mobilization, has been identified as a possible reason why low-fat, high-carbohydrate diets do not result in weight loss for certain individuals.

A study conducted by researchers in Spain supports the thesis that genetic susceptibility to obesity might be modified not only by polymorphisms of genes that regulate lipid metabolism, but also by macronutrient intake and the effects on the phenotypic expression of these "obesity genes." When researchers examined genetic and dietary data collected from 313 Spanish subjects, the results were not as clean-cut as might have been expected. Among the women, those with the Gln27Glu polymorphism were 2.5 times more likely to be obese-but only if their carbohydrate consumption represented over 49% of total energy intake. Those women in the lower half of reported energy intake from carbohydrate who carried the polymorphism were not at increased risk for obesity.

Researchers from the University of Navarro in Spain recruited 313 subjects (66 men, 247 women) between the ages of 20 and 60 to take part in the study. Height and weight measurements were obtained and BMI (kg/m^2) was calculated. Participants were assigned case or control status based on BMI, cases having a BMI > 30 kg/m^2 and control participants having a BMI < 25 kg/m^2. Dietary intake was estimated using a validated food frequency questionnaire and evaluated using Spanish food composition tables. Information on physical activity was obtained and the presence of ß2-AR gene polymorphism determined for each patient.

Out of the 313 participants, 159 were classified as obese (BMI 37.7 ± 5.3 kg/m^2) and 154 as normal-weight (BMI 22.0 ± 1.8 kg/m^2). The Gln27Glu polymorphism was found in 16% of obese and 14.3% of normal-weight participants, indicating that the mutation is not uncommon within this population. Although the presence of Gln27Glu was not directly related to the incidence of obesity, after controlling for age and physical activity, an increased risk for obesity (OR = 2.56, P = 0.051) was found in women carrying the polymorphism whose carbohydrate intake was greater than the median intake (49% of energy) for the group. This observation was not duplicated in the male participants, perhaps because of the small number of men in the study (n=66). A high ratio of carbohydrate to fat intake (CHO/fat > 1.77) was also associated with an increased risk of obesity for female carriers of the polymorphism (OR = 3.21, P < 0.02). In addition, high carbohydrate intake among these women was associated with higher plasma insulin levels, indicating a hyperinsulinemic response.

The results of this study are supported by those of an investigation undertaken in the same Spanish population which found that participants carrying the Gln27Glu polymorphism were less responsive to exercise-induced weight loss, possibly due to a decreased ability to utilize fat stores for energy following physical activity. This study demonstrates that mutations of ß2-AR and other genes that influence lipid metabolism may become clinically important as the influence of gene-environment interactions on the way individuals respond to dietary patterns is clarified. Much more research needs to be completed before individual recommendations can be made. However, awareness of individual variations in diet and exercise response should prompt clinicians to individualize recommendations, monitor progress more frequently, and modify their guidance as needed to improve outcomes.

Martínez JA, Corbalán MS, Sánchez-Villegas A, et al. Obesity risk is associated with carbohydrate intake in women carrying the Gln27Glu ß2-adrenoceptor polymorphism. *J Nutr* 2003;133:2549-2554.

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

Does the Traditional Greek Diet Prolong Life?

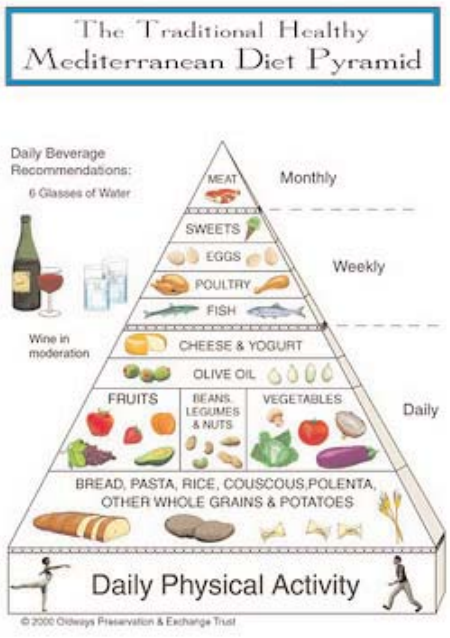
"Olive oil"

A quick word association would take the contemporary lay person about 3 seconds to come up with the "Mediterranean diet." First brought to public attention in the 1950s with Ancel Keys' Seven Countries Study, the Mediterranean diet has inspired endless commentary—and has brought olive oil to the American table. This moderate-fat, moderate-carbohydrate diet that stresses dietary variety and physical activity has been fairly non-controversial through decades of turbulent diet battles. It seems that health professionals and consumers alike are quite amenable to the idea that relatively simple lifestyle choices—eating a variety of fruits, vegetables, nuts, and legumes, letting monounsaturated fats take the place of some favorite foods rich in saturated fat, and making time for moderate daily physical activity—can significantly impact health.

The Mediterranean diet has long been associated with longevity and decreased CHD risk. While the cardioprotective qualities of the Mediterranean diet have been demonstrated in several clinical studies, including the well-known Lyon Diet Heart Study, little research has addressed whether longevity is influenced by long-term adherence to the dietary pattern. Researchers from the University of Athens Medical School, in conjunction with the Harvard School of Public Health, set out to evaluate whether the Mediterranean diet actually does lengthen life.

The researchers recruited 22,043 Greek men, ages 20-86, for participation in a prospective trial that would evaluate the relationship between the degree of adherence to the traditional Mediterranean diet and all-cause mortality. All men were free from CHD, diabetes mellitus, and cancer at baseline. The authors developed and validated a 150-item semiquantitative food-frequency questionnaire, which participants were asked to complete for the year previous to study enrollment. The questionnaire included foods and beverages commonly consumed in Greece and was analyzed using a modified culture-sensitive food composition data base. Intake of the following foods was recorded to determine each participant's degree of adherence to the traditional Mediterranean diet and to control for other dietary factors: Potatoes, vegetables, legumes, fruits, nuts, dairy products (mainly cheese and yogurt), meat, fish, eggs, monounsaturated lipids (mainly olive oil), polyunsaturated lipids (mainly vegetable seed oils), saturated lipids, margarines, sugar, sweets, and nonalcoholic beverages.

The Mediterranean diet is characterized by a low ratio of dietary saturated to monounsaturated fat as well as by the variety of fruits, vegetables, beans, nuts, and legumes that make up a substantial portion of the wider half of its pyramid (see illustration). The diet of each participant was rated with regard to how closely it matched the Mediterranean diet profile and was assigned a score from 0-9, nine being the most similar to the traditional Mediterranean diet. Total energy intake, along with frequency and duration of physical activity, were also estimated (energy expended per kg body weight per day) for inclusion in the statistical analysis.



Adherence to the Mediterranean diet was inversely related to all-cause mortality, independent of sex, smoking status, level of education, BMI, waist-to-hip ratio, and physical activity level. A two-point increase in the Mediterranean diet adherence score was associated with a 25% decrease in total mortality. Of the individual diet components, only fruit and nut intake, and the ratio of monounsaturated to saturated fatty acids were statistically significant predictors of total mortality. A higher degree of adherence to the Mediterranean diet was positively associated with physical activity, but was not related to BMI. Adherence to the diet appeared to decrease the risk of death from both CHD and cancer, although the data were stronger in favor of protection from CHD mortality. It would seem, based on these data, that the best diet for overall health is not necessarily low-fat or low-CHO, but one that is balanced, varied, and moderate.

Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. *N Engl J Med* 2003;348(26);2599-2608.

Plant-Based Diet Stands Up to Statins

New research suggests that a plant-based "portfolio" diet can be as potent as statin drug treatment in improving plasma lipid profiles in hypercholesterolemic adults. Researchers compared changes in serum lipid levels and C-reactive protein levels after one month of either a diet very low in saturated fat plus lovastatin treatment or dietary modification including a "portfolio" of foods shown to be effective in lowering cholesterol levels—plant sterols, viscous fibers, soy protein, and nuts.

Participants included 25 men and 21 postmenopausal women with high serum cholesterol levels at baseline. After a 4-week period in which participants followed a standard therapeutic low-saturated-fat diet, they were randomized to one of three treatment groups:
1) Low-saturated-fat diet emphasizing whole-grain cereals and low-saturated-fat dairy products (n=16; control); 2) Control diet plus 20mg/day lovastatin treatment (n=14); 3) Diet portfolio including plant sterols, soy protein, viscous fibers, and almonds (n=16).

All three diets were similar in saturated fat and cholesterol content. The fatty acid profiles for the portfolio and control diets were also similar. The portfolio diet provided 1.0 g of plant sterols, 9.8 g viscous fibers (from oats, barley, and psyllium), 21.4 g soy protein, and 14 g almonds per 1000 kcals. One hundred grams of okra and 200 g of eggplant were also provided to participants on alternating days as additional sources of viscous fiber. Participants on the portfolio diet consumed 1egg per week and 9 g of butter per day to make the saturated fat and cholesterol content similar to that of the control diet.

At baseline, there were no differences between groups in serum lipids or C-reactive protein. Following four weeks of treatment, changes in serum lipids and C-reactive protein levels were surprisingly similar between treatment groups. LDL cholesterol levels had dropped an average of 30.9% (P<0.001) within the statin group. The portfolio diet group had experienced a parallel decrease in LDL cholesterol of 28.6% (P<0.001). For the statin group, the LDL-HDL ratio had also dropped 28.4% (P<0.001), while the portfolio diet resulted in a 23.5% (P<0.001) reduction. C-reactive protein levels also decreased in both treatment groups- by 33.3% (P=0.002) and 28.2% (P=0.02) for the statin and diet portfolio groups, respectively. Additionally, both the statin treatment and the diet portfolio treatment similarly reduced calculated CHD risk by 24.9% (P<0.001) and 25.8% (P<0.001), respectively.

It is important to note that the control group also experienced a decline in serum lipid and C-reactive protein levels, perhaps due to tightened control of the therapeutic low-saturated-fat diet once the study began. The changes observed in the treatment groups were greater, however, than those experienced by the control group (P<0.005) for LDL cholesterol levels, LDL-HDL ratio, and the calculated CHD risk. Based on these results, it appears that very-low-saturated-fat diets rich in plant sterols, soy protein, viscous fibers, and almonds may be beneficial for hypercholesterolemic adults. However, the sheer volume of high-fiber grains, vegetables, legumes, and soy foods could make the portfolio diet a very difficult therapy to implement among meat-and-potato minded Americans. The portfolio diet contains no meat or dairy products and relies on soy-based foods as a predominant source of protein. It is questionable whether a significant number of statin candidates could be motivated to choose such a diet regimen over drug therapy.

Jenkins DA, Kendall CC, Marchie A, et al. Effects of a dietary portfolio of cholesterol-lowering foods vs. Lovastatin on serum lipids and C-reactive protein.*JAMA*2003;290:502-510.

Antioxidant Vitamins Improve Endothelial Function in Children

Heart disease is no longer just a concern of old age. Although the health consequences of atherogenesis often culminate in mid- to late-adulthood, scientists are discovering evidence showing that its roots can be firmly established in youth. As researchers learn more about atherogenesis, they are beginning to understand that certain aspects of the disease process begin in childhood. For example, the ARMY study (designed to identify and assess cardiovascular risk factors and atherosclerosis in young Austrian males) recently found that over 90% of 17-18 year-old candidates for military service had two or more risk factors for heart disease. The number of risk factors present for each individual was positively associated with intima-media thickness (IMT), a measure of early atherosclerosis. In an effort to determine whether atherosclerosis can be attenuated by means of diet during the childhood and adolescent years, Engler and colleagues examined the individual and combined effects of the NCEP-II diet and supplementation of vitamins C and E on endothelial function, an early predictor of coronary artery disease (CAD) risk.

Fifteen children (7 girls, 8 boys), ages 9 to 20, with familial hypercholesterolemia (FH, n=6) or familial combined hyperlipidemia (FCH, n=9) were recruited to participate in this six-month, double-blind, placebo-controlled crossover study. Participants were instructed in the National Cholesterol Education Program Step II diet (NCEP-II), which they were to follow for the entire six-month study

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period. After the first six weeks on the NCEP-II diet, participants were randomly assigned to the vitamin supplement or placebo group for another six weeks. Following this trial, all participants underwent a six-week washout period, after which they switched treatments to undergo another six-week vitamin vs. placebo trial. The vitamin regimen consisted of 250 mg of vitamin C and 200 IU of vitamin E taken twice daily. Endothelial function was assessed by measurement of flow-mediated dilation (FMD) of the brachial artery at baseline and every six weeks thereafter. Blood and urine samples were also obtained every six weeks to measure biomarkers of oxidative stress and inflammation (C-reactive protein).

Endothelial function was impaired at baseline for this study group. Adherence to the NCEP-II diet alone resulted in an 8% reduction in LDL-cholesterol levels. By study completion, there were no changes in any other lipid levels or in FMD as a result of diet or antioxidant therapy. Biomarkers of oxidative stress and inflammation remained unchanged throughout the study for both intervention groups. Although the six-week antioxidant supplementation period resulted in no changes in blood lipid profiles, markers of oxidative stress, or inflammation, this regimen resulted in significant improvements in FMD from baseline (5.7 ± 2.9% to 9.5 ± 4.2%; $P<0.001$), indicating that these antioxidants were instrumental-either independently or synergistically-in improving endothelial function.

The results of this study indicate that for children with FH or FCH, supplementation with vitamin C and vitamin E in addition to diet therapy might be effective in reducing long-term CHD risk by improving endothelial function. The short duration of this study, however, limits immediate application of its results. The absence of any effect of the NCEP-II diet on FMD measurements would suggest that there are no acute effects of the diet as were seen during the vitamin supplementation period. Whether the long-term effects of the two treatments on FMD are different or similar following years of intervention is still unknown. Until long-term studies are completed, it cannot be determined which of the treatments, or combination of treatments, actually reduces CHD risk when initiated during childhood.

Engler MM, Engler MB, Malloy M, et al. Antioxidant vitamins C and E improve endothelial function in children with hyperlipidemia: endothelial assessment of risk from lipids in youth (EARLY) trial. *Circulation* 2003;108:1059-1063.

Knoflach M, Kiechl S, Kind M, et al. Cardiovascular risk factors and atherosclerosis in young males: ARMY study (atherosclerosis risk-factors in male youngsters). *Circulation* 2003;108-1064-1069.

Early-Childhood Diet Intervention Might Reduce Risk of CHD

With the growing problem of obesity among American youth, it should not be surprising that an alarming number of school children are showing early signs of vascular disease. Results from a recent study presented at the American Heart Association's Scientific Sessions 2003 indicate that an estimated one in eight children between the ages of 8 and 17 has at least three risk factors for the metabolic syndrome, a precursor of CVD. These risk factors include high blood pressure, high triglycerides, low HDL-cholesterol levels, glucose intolerance, elevated insulin levels, and excess body weight. Furthering the evidence that atherogenesis may begin in the first decades of life, research by Knoflach et al. has recently demonstrated that multiple risk factors for atherosclerosis are associated with increased intima-media thickness (IMT), an indicator for the presence of atherosclerosis, in adolescent boys. Researchers speculate that stopping or slowing the progress of early atherogenesis might be key to preventing the later onset of CHD. To address this possibility, Kaitosaari et al. followed a large cohort of Finnish infants for seven years to evaluate the impact of a low-saturated fat diet on serum lipoproteins.

The study began in 1990, when 1062 healthy infants were enrolled and randomized to a diet intervention (n=540) or control group (n=522). Blood samples were collected when infants reached their 7th month of age to establish baseline data. Parents of infants assigned to the intervention group were instructed in feeding their children a diet consistent with the Nordic Dietary Recommendations (10-15% energy from protein, 30% energy from fat, 55-60% energy from carbohydrate; ≤10% energy from saturated fat). Parents of infants in the intervention group were instructed in diet and lifestyle interventions twice a year and were asked to provide 4-day food records for their children at the same intervals. A total of 511 children finished the 7-year study. At 7 years, blood samples were analyzed for serum total, HDL, and non-HDL cholesterol levels, along with apoA-I, apoB, and triglyceride levels. Additionally, 197 children were randomly selected for testing of HDL and LDL subfractions to evaluate particle size.

Statistically significant changes were observed only in male participants. At 7 years, the intake of saturated fat for the male intervention group was 11.5% of energy, compared to 13.6% of energy for the male control group. The mean plasma total cholesterol level for the intervention boys was 5% lower than for control boys, while the mean LDL cholesterol level was 7% lower and the apoB concentration 9% lower for the diet intervention vs. control group. The intervention boys also had

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more favorable HDL/total cholesterol ratios by 5%. No association was observed between carbohydrate intake and serum lipid values for boys or girls.

HDL and LDL subfraction testing was completed for a total of 81 boys and 95 girls. No differences were detected between control and intervention groups of either gender for HDL subfractions. Similar to the serum lipid evaluations previously described, no differences were seen between control and intervention girls for LDL subfractions. Among the boys, however, average major LDL particle diameter was larger for those in the intervention group vs. control group (263 Å vs. 257 Å). Larger LDL particles have been associated with lower risk for CHD, while smaller, denser LDL particles are thought to be more atherogenic due to lower LDL receptor binding affinity, lower resistance to lipid peroxidation, and ease of entrance into arterial walls.

The authors conclude that initiating a low-saturated-fat diet early in life might help slow atherogenesis for boys. This risk reduction is thought to be achieved not only by an effective reduction of total and LDL cholesterol levels, but also by modification of the size, and thus the atherogenicity, of LDL particles. What effects the intervention might have on other aspects of the metabolic syndrome remain unknown. The differential effects of the intervention in boys vs. girls raises interesting questions regarding whether there is a need to develop specific dietary interventions for lowering CHD risk in young females.

Kaitosaari T, Rönnemaa T, Raitakari O, et al. Effect of 7-year infancy-onset dietary intervention on serum lipoproteins and lipoprotein subclasses in healthy children in the prospective, randomized special Turku coronary risk factor intervention project for children (STRIP) study. *Circulation* 2003;108:672-677.

Knoflach M, Kiechl S, Kind M, et al. Cardiovascular risk factors and atherosclerosis in young males: ARMY study (atherosclerosis risk-factors in male youngsters). *Circulation* 2003;108:1064-1069.

Editorial

Guilty and Not Allowed to be Proven Innocent

What a terrible way to spend the holidays! I am really getting tired of running up and down the stairs and back and forth from room to room. Four court cases at one time is certainly no way to start 2006. The funny thing is that each case had the same theme - someone decides you're bad and that you do terrible things, they righteously tell everyone with grand embellishment just how bad you are, and then they gleefully sue you for the misinformation you put out to defend yourself from their "truth." In one court room sat CSPI with a suit against the egg industry for inferring that eggs were not cholesterol-laden killers but something that actually contributed to nutrition. In another court PETA was claiming that the egg industry's use of science-based animal welfare standards was insufficient and we had no right to make misleading claims that we treat birds humanely. Upstairs Greenpeace was suing with the usual confrontational charge of "too little, too late" over promotion of industry guidelines on environmental programs. And finally, John Banzhaf and his "gouge'em and sue'em" colleagues were suing "Big Egg" for something, although no one was exactly sure for what, given his convoluted concepts of industry being liable for everything. All this legal reactivity because the egg industry quit being a doormat for every animal rights-vegetarian-environmental nut case and paternalistic do-gooder who felt a compulsion to accuse it of everything from death and taxes to the demise of western civilization. Of course we all know that the real goal here is a relentless push towards a vegetarian society with no animal agriculture. Eggs are just one of many such targets these groups have chosen.

Seems some folks are allowed to picket, demonstrate, put up billboards, print outrageous ads (like the "Holocaust on Your Plate" project), say whatever non-science nonsense they want to the media, throw dead chickens or manure on industry representatives, break into and damage production facilities, and in general make themselves extreme pains in the posterior—and still be considered honest and virtuous and noble. This of course gets them more than their fair share of media attention as they express their First Amendment rights. But just let industry say, "wait a minute, that's not accurate, that's not true," and it is tagged as self-serving and profit-motivated. Then industry easily ends up in court trying to prove that its rebuttals to the false accusations are true and factual and not misleading. I wonder why the loud mouths starting it all aren't required to prove their charges in the first place. It's like, "I can lie about you but you're not allowed to tell the truth about me."

This all started a few years back with a suit against Nike's attempts to address accusations of sweat shop labor practices, which Nike lost. The strategy became very simple and oh so easy to use for jamming the courts and thwarting industries. The sequence of events are easy to follow and make two groups very happy: anti-something/anything activists and trial lawyers. First, activists attack an industry via media, internet, demonstrations, etc. Second, the industry can either stay silent (making them clearly guilty) or respond in self-defense. Third, activist lawyers take the industry to court on charges of making false or misleading "commercial statements" (i.e. false advertising) to the public. And of course, in addition to the punitive damages requested in the lawsuit, the plaintiff includes all "reasonable attorneys' fees and court costs." So the system allows unrestricted freedom of speech for those attacking an industry, and restricted "commercial speech,"—with a

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very high standard of proof—for the industry to use in its defense. And just to be perfectly clear, this can extend far beyond advertising to include educational materials, web site contents, letters and announcements sent by the industry, and, as I found out, any formal scientific presentation. [Just ask the Colonel about web site content and lawsuits.] Seems easier to get a health claim than to say you're not harmful.

It's really an old ploy with a new twist where you ask a question and no matter what you answer you win. Questions like: Have you stopped selling cholesterol-laden pellets to the public? Have you stopped mistreating and tormenting your chickens? Have you stopped polluting the waterways and air? Answer yes and it means you were guilty of doing it in the first place, answer no and it means you're still doing it. Damned if you do, damned if you don't.

Oh well, I have to get back to the courtroom. Since my name was included on all the lawsuits, I spend a lot of time in courtrooms these days, and in the bank getting funds to pay for this folly. Two successes at once for the shrill voices of doom and gloom out there: a stymied promotion program spending it's time and money on court cases, and a financial drain on those who they think profit from their evil deeds. What is really dispiriting is that these tactics do have an effect on an industry, and eventually that hits the consumer because we all know who eventually ends up paying for the trial lawyers and for the activists' "I hate progress" programs - you and me. But right now I'm paying twice!

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