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I N S I D E

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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.

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

Pediatric Egg Intake and CAD Risk Modification

It is well known that CAD risk varies between ethnic groups. The observed disparity in CAD risk between geographic and ethnic populations is thought to be due partly to genetic predisposition and partly to environmental modifiers such as diet and physical activity. Having a high prevalence of insulin resistance and type 2 diabetes, the population of northern Mexico is particularly susceptible to CAD. The extent to which dietary intake might influence CAD risk in this population is not known, however, the implications of research examining how modifiable risk factors (such as diet and exercise) might alter lipid profile and LDL atherogenicity, and thus alter individual predisposition to CAD, are critically important. It is also unknown whether modifiable risk factors in childhood contribute to later CAD risk. To better understand the impact of dietary intake (particularly dietary fat and cholesterol) on modifiable risk factors during childhood, Ballesteros et al. examined the influence of adding dietary cholesterol from eggs to the baseline diet of a cohort of children from northern Mexico. In a short-term, randomized, controlled study, Ballesteros et al. demonstrated that egg intake among a cohort of 54 youth aged 8-12, at high risk for CAD, did not increase CAD risk.

Sixty children (30 boys, 30 girls) aged 8-12 years were recruited from a low socioeconomic area of Hermosillo, Mexico. Participants were randomly assigned to the egg (EGG) group or egg substitute (SUB) group. Those in the EGG group consumed 110 g (or the equivalent of two whole eggs) for 30 days, adding 518 mg cholesterol to their usual daily intake. Those in the SUB group consumed an equivalent weight of egg whites for 30 days, adding no cholesterol to their usual intake. Participants were instructed to follow their usual diet over the course of the study. Following the initial 30-day diet period and a 3-week washout period, participants switched to the alternate diet regimen for an additional 30 days. Eggs and egg substitute meals were served to the children in the school cafeteria during breakfast. Parents were responsible for egg feeding over the weekend. Compliance was >98% both during the week and on the weekend. The children wore pedometers for seven consecutive days at baseline and during both treatment periods to evaluate changes in physical activity over the course of the study. The researchers utilized three-day weighed food records to evaluate the nutrient composition of participants' diets.

Two fasting blood samples were taken on separate days prior to initiation of the study to determine baseline lipid

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profiles for each participant. Fifty-four children completed the study (25 boys, 29 girls). An increase of 0.05-0.06 mmol/L (1.9-2.3 mg/dL) in total cholesterol can be expected with the addition of 100 mg/d dietary cholesterol. Therefore, children whose total cholesterol increased > 0.06 mmol/L for each additional 100 mg cholesterol per day were categorized as hyperresponders. All others were considered hyporesponders. (Editor's comment: Since the cholesterol intakes were not adjusted for differences in body weights and cholesterol absorption values are unknown, the classification of hypo- and hyperresponders is somewhat arbitrary.)

Baseline plasma lipids, physical activity level, blood pressure, and BMI were similar for both boys and girls later classified as hypo- and hyperresponders. The only significant difference between boys and girls at baseline was BMI.

The usual diet for all participant children was found to be high in fat and saturated fat (37-43% and 10.5-13.6%, respectively) and did not differ between diet periods. Dietary cholesterol intake was significantly higher during the EGG period, as expected. No differences were observed between diet periods with regard to physical activity, systolic blood pressure, or BMI. Diastolic blood pressure was lower during the EGG period for both hypo- and hyperresponders ($P<0.05$). Although hyperresponders experienced a significant increase in both LDL (from 59.4±14.7 to 74.5±13.9 mg/dL) and HDL cholesterol (from 47.5±10.0 to 52.1±11.2 mg/dL) during the EGG period, the total cholesterol:HDL and LDL:HDL ratios did not change significantly, indicating that CAD risk did not increase for this group with increased egg intake. Neither LDL, nor HDL cholesterol concentrations differed significantly from baseline for hyporesponders following the EGG period or the SUB period. Plasma apo B concentrations were similar for hyper- and hyporesponders and did not change based on diet group assignment. Apo B concentrations did not change with the change in LDL cholesterol concentrations, indicating that LDL particle size, not number, increased for hyperresponders. Interestingly, of the 34 children that were classified as having pattern B LDL particles during the SUB period, 5 shifted to pattern A (larger, less-dense LDL particles) during the EGG period.

Because the children's activity levels and body weights remained constant throughout the study, it is probable that the dietary treatment was the variable that had the largest impact on plasma lipids. Adding dietary cholesterol from eggs to the diets of these children did not increase biomarkers for CAD risk. Conversely, it resulted in less-atherogenic LDL particles and an increase in HDL cholesterol concentrations that offset the increases in LDL cholesterol. The researchers conclude that intake of dietary cholesterol from eggs in this high-risk pediatric population does not negatively affect biomarkers for CAD risk.

Ballesteros MN, Cabrera RM, Saucedo MS, and Fernandez ML. Dietary cholesterol does not increase biomarkers for chronic disease in a pediatric population from northern Mexico. *Am J Clin Nutr* 2004;80:855-61.

Note: This study was funded by a grant from the American Egg Board administered by the Egg Nutrition Center.

Key messages

- In children whose plasma LDL cholesterol levels increased with egg feeding, there was a compensatory increase in HDL cholesterol that left the total cholesterol:HDL and LDL:HDL ratios unchanged. The apo B concentration did not change following egg feeding for these children, indicating an increase in the size, rather than in the number of LDL particles.
- Adding cholesterol from egg yolks to the diets of young children predisposed to CAD did not increase biomarkers for CAD risk.

DHA and Visual Acuity in Infants

Breastfeeding has been associated with health benefits related to nutrition, cognitive development, and immune function, among others. Intake of breastmilk appears to be particularly important in visual development during infancy. Research indicates that visual function is more advanced in breastfed vs. formula-fed infants. Although the exact mechanism behind this observation remains unknown, research suggests that the long-chain PUFA, docosahexaenoic acid [22:6(n-3), DHA], is primarily responsible. DHA has been directly associated with improved visual and mental development. Since breastmilk is the major source of DHA for infants (and because weaning foods are generally poor sources of DHA), plasma levels of this long-chain PUFA generally decrease at about 6 months when infants begin the weaning process. It is not known whether supplementation of DHA during weaning further benefits visual development. To address this question, Hoffman et al. designed a randomized clinical trial to evaluate whether DHA-enriched baby food provided to infants during weaning would enhance visual development.

Fifty-five infants who had been exclusively breastfed from birth were randomly assigned at 6 months of age to one of two study groups. Infants in the DHA group received baby food made with DHA-enriched egg yolks. Infants in the second group received control baby food, which contained no DHA-enriched egg yolks, but was made from the same ingredients otherwise. Parents were instructed to give their infant one jar of study baby food per day for a period of 6 months. Assessments of growth (weight, length, head circumference, and triceps and subscapular skinfold thickness) and two measures of visual development [general acuity and stereoacuity] were done at 6, 9, and 12 months. Blood samples were also analyzed at 6 and 12 months.

Fifty-one infants completed the study, 26 in the control group and 25 in the DHA group. No differences existed between the groups with respect to RBC lipid composition at baseline (6 months). However, at 12 months, RBC DHA levels had decreased for the control group from 3.8% to 3.0% (P=0.012) and had increased in the DHA group from 4.1% to 5.5% (P<0.002). With regard to visual development, both groups of infants demonstrated improved general acuity from 6 to 12 months, as expected. However, the DHA supplemented infants improved more than controls by approximately 1.5 lines on an eye chart (P<0.002). General acuity measurements correlated strongly with RBC DHA levels at 12 months (r=-0.50; P=0.0002), further indicating that DHA supplementation accelerated the maturation of visual acuity in these infants. Stereoacuity also improved for both groups, but did not differ between groups at 12 months. Despite the higher fat and calorie content of the DHA supplemented baby foods (5-6 times the fat content and ~230 kJ/100g more calories than control food), infants in the control and DHA groups did not differ with regard to growth parameters. Length, weight, head circumference, and skin fold thicknesses were similar between groups at 6, 9, and 12 months (P>0.3).

This research demonstrates that infants exclusively breastfed from birth to 6 months who received baby food containing DHA-enriched egg yolks during weaning had higher RBC DHA levels and accelerated development of general acuity compared to controls. Although the DHA-supplemented infants appeared to have no advantage over non-supplemented infants with regard to stereoacuity, the difference in general acuity was approximately equivalent to 1.5 lines on an eye chart. The authors conclude that DHA intake remains important during and beyond the weaning period in the first year of life, when intake of breastmilk decreases.

Hoffman DR, Theuer RC, Castañeda YS, et al. Maturation of visual acuity is accelerated in breast-fed term infants fed baby food containing DHA-enriched egg yolk. *J Nutr* 2004;134:2307-2313.

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

Low-Fat vs. High-MUFA Diets in Type 2 Diabetes

The past century has seen significant changes in diet recommendations for individuals with diabetes. Diabetic diet recommendations have, of necessity, focused first on glycemic control, but a second priority has asserted its importance in recent decades—that of preventing coronary artery disease. The advent of insulin ended the requirement for strict ketogenic diets and allowed the diabetic diet to include carbohydrates. It was postulated that a high-carbohydrate, low-fat diet would help decrease plasma cholesterol in diabetic individuals, however, this approach also has the potential to increase triacylglycerol concentrations and to complicate glycemic control.

Replacing some carbohydrate with monounsaturated fat has been suggested as a means of avoiding the atherogenic effects of high carbohydrate intake while maintaining a diet low in saturated fat and cholesterol. Research supporting this hypothesis led to a recommendation from the American Diabetes Association that a combination of carbohydrate and monounsaturated fat should make up 60-70% of total energy. However, research supporting this recommendation was carried out using controlled metabolic studies in which diabetic participants' dietary intakes were tightly monitored. It is not known whether diets high in monounsaturated fats are beneficial in free-living diabetic populations. To evaluate this recommendation, Gerhard et al. compared ad lib low-fat (emphasizing fiber and complex carbohydrates) and high-monounsaturated fat diets in a diabetic population.

Eleven individuals (8 women, 3 men) with type 2 diabetes were recruited to participate in this cross-over study. All participants had good glycemic control prior to initiation of the study, with an average glycated hemoglobin (HbA_{1c}) of 6.8±1.0. None were being treated with insulin therapy. Participants were randomly assigned to either a low-fat (LF) or high-monounsaturated fat (MUFA) diet for 6 weeks, after which they underwent a 6-12 week washout period and followed the alternate diet for an additional 6 weeks. All meals were prepared in a metabolic kitchen and one meal per day was consumed at the research center. Remaining meals were packaged and sent home with participants. Because the study objectives called for ad libitum consumption, participants were provided with enough food to cover their calorie needs plus 25% and were instructed to eat each meal to satiety and return the uneaten portion.

The LF diet consisted of 20% of calories from fat, 65% from carbohydrates, and 10% from refined sugar. It provided more fiber and water, weighed more, and was less calorie-dense than the high-MUFA (HM) diet. The LF diet was also lower in saturated fat and cholesterol than the HM diet. (Comment: It was presumed by the investigators that in general, ad lib HM diets are higher in saturated fat and cholesterol than their LF counterparts. However, this is debatable since most common sources of monounsaturated fat—olive oil, peanut butter, almonds, avocados—are low in saturated fat and contain no cholesterol.)

Participants took in fewer calories during the LF diet period than during the HM period ($P<0.02$), consuming an average of 212 fewer kcals per day. The LF diet intervention resulted in an average differential weight loss of 1.06 kg, consistent with what would have been expected with the reported calorie deficit. Weight loss during the HM period was not statistically significant.

No differences were observed between diets with regard to plasma lipoprotein levels. Total, HDL, and LDL cholesterol concentrations had decreased from baseline following both diets, but the ratios of LDL:HDL and total:HDL cholesterol remained the same. Neither plasma triacylglycerol nor VLDL cholesterol concentrations changed as a result of dietary intervention. Glycemic control did not differ significantly between diet interventions.

The researchers conclude that ad lib consumption of a low-fat, high-fiber, high complex-carbohydrate diet, contrary to many previous studies, might be more effective than ad lib high-monounsaturated diets in inducing weight loss for individuals with type 2 diabetes. They speculated that the calorie deficit observed between the two interventions was partially due to the lower energy density of the low-fat regimen, which included foods low in calories and higher in water and fiber. The sample size for this study was small (n=11). The results warrant further research in a larger subset of the diabetic population and potentially with an ad lib HM diet lower in saturated fat and cholesterol.

Gerhard G, Ahmann A, Meeuws K, et al. Effects of a low-fat diet compared with those of a high-monounsaturated fat diet on body weight, plasma lipids and lipoproteins, and glycemic control in type 2 diabetes. *Am J Clin Nutr* 2004;80:668-73.

Oats, Antioxidants, and Postprandial Endothelial Function

Endothelial dysfunction, or the inability of arteries to respond appropriately to stressors on vessel walls, is strongly associated with the development of coronary artery disease (CAD) and its risk factors. Efforts to reduce risk factors for CAD generally result in improved endothelial function and it is generally accepted that such improvement indicates reduction in CAD risk. Soluble fiber such as that found in oats has been shown to decrease CAD risk by lowering total and LDL cholesterol concentrations as well as blood pressure in hypertensive individuals. Acute endothelial dysfunction following fat ingestion is thought to increase the risk of acute coronary events in susceptible individuals. Ingestion of oat fiber has been shown to ameliorate the effects of acute fat ingestion on postprandial endothelial function and it is thought that the addition of antioxidants might provide further benefits. To examine the separate and combined effects of oat fiber and antioxidants on acute endothelial function (EF), Katz et al. undertook a randomized, blinded, crossover trial using oats and antioxidant vitamins C and E.

Thirty (30) subjects (16 male, 14 female) were recruited to participate in this trial. Men were between the ages of 35 and 75. Women were postmenopausal, but not over 75 years of age. All had normal cholesterol and triacylglycerol concentrations and BMI <25. All were nonsmokers and had no known existing CVD.

The trial was undertaken in two phases. The first phase was designed to measure the acute effects of oat and antioxidant consumption on EF. Participants underwent brachial artery reactivity scans (BARS) once each week over 4 consecutive weeks to test their arterial responses (flow-mediated dilation [FMD]) to acute ingestion of a fatty test meal with one of four treatments (60 g rolled oats, 500 mg vitamin C + 400 IU vitamin E, oats + vitamins, or placebo). A baseline BARS was completed in the morning, prior to treatment, and was repeated 3 hours postprandially for each participant and with each treatment.

The objective of the second phase was to evaluate the effects of sustained oat and antioxidant consumption on EF. Participants were randomly assigned to one of the four daily treatments for a 6-week block, after which they underwent pre- and postprandial BARS testing and a 2-week washout period before beginning the next 6-week treatment phase. This was repeated until all participants had undergone each treatment regimen. Participants on the oat treatment consumed 60 g of oatmeal for breakfast each day and an oat bran cereal snack each afternoon (for a total of approximately 5 g of β-glucan). The vitamin treatment was as described above (500 mg vitamin C + 400 IU vitamin E).

Twenty-six participants (15 male, 11 female) completed both phases of the study. In the acute phase, FMD increased nonsignficantly from baseline following oat ingestion, and decreased nonsignificantly from baseline following treatment with vitamins + oats and vitamins alone. Results were similar following six weeks of dietary treatment. FMD increased from baseline with sustained oat treatment and the vitamins + oats and vitamins alone resulted in decreased FMD. These observations were, again, nonsignificant and the treatments did not differ significantly from each other with respect to FMD. When data from the two study phases were pooled, oat intake was associated with a 3% increase in FMD as compared with the vitamin treatment, which resulted in a 1.6% decrease (p=0.04) and the vitamins + oats treatment, which resulted in 1.2% decrease in FMD (p=0.02).

This research suggests that consumption of oats might help improve postprandial EF following acute fat ingestion, but it does not support the use of antioxidant vitamins C and E for this purpose. When vitamin C and E were added to the oat treatment, FMD worsened nearly as much as when vitamin C and E were used alone. The reason for this is not clear. Perhaps a larger sample size would have given statistical significance to the outcomes observed in this study, however their biological significance would not be known since the observed changes in FMD were relatively small. Since vitamins C and E had an apparently adverse influence on FMD in this study, more research is needed to clarify the effects of antioxidant vitamins on postprandial endothelial function.

Katz DL, Evans MA, Chan W, et al. Oats, antioxidants and endothelial function in overweight, dyslipidemic adults. *J Am Coll Nutr* 2004;23(5):397-403.

Editorial: *Death of a Theory—The Five Stages of Scientific Grief*

Twenty-five years ago the Egg Nutrition Center was established by the egg industry to question (and hopefully to refute) the widely held theory of a positive relationship between eggs, dietary cholesterol, blood cholesterol, and heart disease risk. Since 1979, the Egg Nutrition Center has been successful in poking some pretty big holes in the hypothesis, and in many parts of the world the dietary cholesterol-heart disease relationship is now lifeless and discarded. Having watched this development over the past twenty years I've been struck by the similarities between the demise of a cherished scientific theory and the five stages of grief. If, during the tedious death of a long held hypothesis, its "survivors" get stuck in one stage or another, the process of grieving (i.e. theory correction) cannot be completed, and the invalid theory persists as long as those grieving for it will not come to terms with the facts and move on.

The first stage of grief is **denial**. This is often evident by withdrawal to avoid facing the loss, avoidance of those people who confront us with the truth, refutation of any contrary evidence, and rejection of those who bring us the unacceptable facts. There are those scientists who will not even acknowledge the possibility that "their theory" might not stand the test of time. As the counter evidence accumulates, they choose to ignore signs that maybe "their theory" isn't valid and needs to be re-examined. The attitude is one of "how dare they try to negate all the good we've accomplished dictating our concepts to the policy makers and the public?" ("*A theory, if you hold it hard enough and long enough, gets rated as a creed....*" Robert Frost (1874–1963), U.S. poet.)

The second stage of grief is **anger**. That feeling of wanting to fight back or get even with colleagues or opponents, and finding ways to reprimand the heretics for challenging the theory. This is often expressed by working to discredit the scientific credentials of non-believers, working to limit grant funds for the disruptive research of trouble makers, keeping the unfaithful off committees and panels where their biased and obviously unfounded viewpoints could be heard, and making sure that they never, ever get on any advisory committee reviewing the evidence (or lack thereof) behind the theory. And above all else, attack their motives and credibility. Surely they must be bought and paid-for industry inspired talking heads to utter such nonsense. ("*... what a weak barrier is truth when it stands in the way of an hypothesis!*" Mary Wollstonecraft (1759–97), British feminist.)

The third stage of grief is **bargaining**. In science this often expresses itself as attempting to justify the seriously wounded theory using any available type of evidence, no matter how weak the data. In the egg-dietary cholesterol-heart disease battles this has gone from saying that an egg a day would raise cholesterol levels by 20 mg/dl and the relative risk of heart disease by 20%, to an egg a day raising cholesterol levels by 5 mg/dl and heart disease risk by 5%, to meta-analyses estimating that an egg a day will raise heart disease risk by 2% (even this latest estimate was found to be seriously flawed). And then one can always rely on the old bromide that "it can't hurt" to limit eggs. ("*It is the nature of an hypothesis, when once a man has conceived it, that it assimilates every thing to itself as proper nourishment; and, from the first moment of your begetting it, it generally grows the stronger by every thing you see, hear, read, or understand.*" Laurence Sterne (1713–68), British author, clergyman.)

The fourth stage of grief is **despair**. Here the proponents of the theory develop an overwhelming sense of hopelessness, frustration, bitterness, and self-pity while they mourn the brutal violation of "their theory" as well as the hopes, dreams and plans of what "their theory" could have been in the future. This sense of despair and betrayal often leads to writing and publishing lopsided and selectively referenced review articles in an attempt to resuscitate the mortally wounded hypothesis, and the re-analysis of ancient and questionable data sets which once protected the theory from scrutiny and hopefully will once again provide the crutches for it to limp along a little longer. These desperate attempts at justification are a clear sign that professional viewpoints have changed and that the all important "consensus of scientific opinion" has begun to disintegrate. ("*The great tragedy of science—the slaying of a beautiful hypothesis by an ugly fact.*" Thomas Henry Huxley (1825–95), British biologist and educator.)

The fifth and final stage of grief is **acceptance**. This stage of the process reaches different people and groups at different rates, some reach it quickly while others dawdle and procrastinate in fear that there are still those out there in the anger stage. For the egg, the stages of acceptance range from Canada not having a Daily Value for cholesterol and eggs carrying the Health Check mark of the Heart and Stroke Foundation of Canada, to the American Heart Association doing away with its thirty year old prohibition of "no more than three whole eggs per week", to the National Cholesterol Education Program still believing that lowering dietary cholesterol from 300 to 200 mg/day actually does something to decrease heart disease risk. The ultimate acceptance is the recognition by many that eggs have nutritional value contributing high quality protein, choline, lutein and a wealth of vitamins and minerals. ("*Science has fulfilled her function when she has ascertained and enunciated truth.*" Thomas Henry Huxley (1825–95), British biologist and educator.)

So after 25 years the scientific community seems to be pretty well spread across the various stages of grief for the theoretical egg-dietary cholesterol-heart disease relationship. I like to think that at

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least a majority are pretty far down the sequence of stages with a large number having reached acceptance of its demise (People always tell me that they never gave up on the egg even with all the accusations laid on its shell). I guess the next stage in the process is the one based on the old adage that theories only really die when their proponents do. And after twenty-five years of working to get the facts out about eggs and health (and experiencing the various aspects of denial, anger, bargaining and despair of the scientific community), the Egg Nutrition Center can look forward to the coming years as a time to advance the many health benefits of including eggs in a balanced diet. I expect that the next 25 years will be easier than the last 25. ("*Science is a cemetery of dead ideas.*" Miguel de Unamuno (1864–1936), Spanish philosophical writer.)

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

First-Course Salads Induce Satiety

Effective strategies to increase satiety, and thus reduce mealtime energy intake, are highly valued in our weight-conscious society. Such strategies should help in containing overall daily energy consumption. One strategy thought to be effective in increasing satiety and decreasing energy intake is incorporating a low-calorie, filling, first course into the main meal. Although many researchers hypothesize that this strategy could help individuals spontaneously reduce energy intake during the main course by increasing fullness and satiety, the argument is that incorporating this extra course would only increase mealtime and overall energy intake. Researchers at Pennsylvania State University undertook a study to evaluate the effectiveness of including a pre-meal salad (of varying size and energy content) in reducing total energy intake during the subsequent meal.

Forty-two women between the ages of 19 and 45 with BMIs between 18 and 35 participated in this randomized crossover study. All women were non-smokers who typically ate three meals a day. None were pregnant, breastfeeding, dieting, or taking medications known to affect appetite. None had any known food aversions, food allergies, avoidances, or disordered attitudes toward eating. Participants were not aware of the study objectives.

Participants ate lunch once a week for seven consecutive weeks in the research laboratory and were randomly assigned to eat one of 6 experimental salads or nothing (control) prior to the meal. The six salads were varied both in energy density (0.33, 0.67, or 1.33 kcal/g) and size (150 or 300 g). Energy density was determined by altering amounts and types of dressing and cheese. Participants were instructed to eat the entire salad prior to beginning their main course of pasta, of which they were allowed to eat as much or as little as desired. Foods were weighed before and after each meal to get an accurate measurement of how much of each course was eaten. The main course was served twenty minutes after the salad was finished. Participants were seated in isolation from each other. They were instructed to keep their evening meals and activity levels consistent for the day before each test day and were required to consume nothing but water between breakfast and lunch on the day of the test. Water was also restricted in the hour before the test meal was given. Water consumption during the meal was ad lib. Participants used hunger and satiety scales to measure their relative hunger and satiety before beginning the meal, after the first course, and following the meal.

Portion size was more important than energy density in determining the calories eaten during the second course. When salad size increased from 150 g to 300 g, subsequent consumption of calories from the pasta course decreased by 98±30 kcals ($P<0.0001$). The effect of energy density of the salads on consumption of the pasta course did not reach statistical significance ($P=0.06$).

Energy density of the salad course was the most important determinant of the total calories eaten during the entire meal. Compared to control (no first-course salad), consumption of the smaller low-calorie salad decreased overall meal intake by 7%, while eating the larger low-calorie salad decreased overall meal intake by 12%. When the smaller portion of the high-calorie salad was eaten, overall energy consumption for the meal increased by 8%. Overall energy intake increased by 17% when the larger portion was eaten. There were no differences between participants' hunger and satiety ratings prior to eating the salad. Independent of energy density, larger salads induced lower hunger ratings and higher fullness ratings than smaller ones ($P<0.0001$ for both).

Results from this study suggest that eating a low-calorie salad before a meal increases satiety, and that eating a larger low-calorie salad results in an even greater satiety effect. Thus, adding a filling, low-energy course to the beginning of the main meal appears to induce a spontaneous decrease in the energy consumed during the meal.

Rolls BJ, Roe LS, Meengs JS. Salad and satiety: energy density and portion size of a first-course salad affect energy intake at lunch. *J Am Diet Assoc.* 2004;104:1570-1576.

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