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INSIDE

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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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Childhood Obesity and CVD Risk

Using data from 8,270, 4 to 12 year olds enrolled in the National Longitudinal Survey of Youth (NLSY), Strauss and Pollack determined the changes in overweight incidence between 1979 and 1998. Like the NHANES III findings, which showed that 14% of US children are overweight, results from the NLSY found that American children are losing the battle of the bulge. This is especially problematic for Hispanic and African-American kids, and children from the Southern states.

Data on a child's height, weight, demographic, and socioeconomic status were collected every 2 years by in-home interviewers. The results showed that, like adults, the prevalence of overweight children had increased dramatically during the study period. For example, compared to 1986, overweight prevalence in 1998 was 120% higher in African-American and Hispanic children and 50% higher in white children. These data indicated that in these populations, there are 12.3% overweight white children, 21.5% overweight African-American children, and 21.8% overweight Hispanic children with BMI values >95% for age and sex. Also, the relative weight of the overweight child increased from 1986 to 1998, indicating that the weight problem is a case of both severity and prevalence.

Detailed analysis revealed that race and geographical location played a strong role in predicting the prevalence of overweight children. For example, African-American and Hispanic children and children from the southern states were more likely to have weight problems than non-Hispanic white children or children from other regions of the country. On the other hand, Strauss and Pollack found that income was less directly associated with weight. Income was inversely associated with weight gain in the white cohort with an odds ratio of 0.78, in the African-American cohort it was directly associated (OR 1.39) and equivocal in the Hispanics with odds ratio of 0.88.

The results reported by Strauss and Pollack indicate that weight problems not only affect adults and adolescents, but is a growing problem for very young children between 4-12 years of age. And the racial disparity in weight gain observed in this study concerned the researchers due to possible long-term racial disparity in health outcomes.

These negative trends of a high prevalence of obesity among children are especially concerning since earlier studies show that obese children are more likely to become obese adults. The Newcastle Thousand Families Study, which followed 1,142 children from birth to age of 50, confirms this theory. The cohort size at the end of the study was 529. According to the researchers,

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Childhood Obesity

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children in the highest BMI group at age 9 or 13 were 5 to 9 times more likely to have a BMI of >30 at age 50 with a regression coefficient of 0.24 ($p<0.001$) and 0.39 ($p<0.001$) at ages 9 and 13, respectively. However, the association between childhood obesity and body fatness at 50 years old was weaker with a regression coefficient of 0.1 ($p=0.07$) and 0.22 ($p<0.001$) at ages 9 and 13, respectively. The data also showed that a majority of adults in the highest percent body fat were once thin children with low BMI's at age 9 and 13. Children in the bottom quarter for BMI were equally likely to be have high body fat as adults, as were the children in the top 10% of BMI at age 13.

An interesting finding from this study was that even though BMI at age 50 was directly related to CVD risk factors, BMI at age 9 and 13 were not related to CVD risk factors. For example, even after adjusting for adult BMI, childhood BMI showed an inverse association with CVD risk. In women, fasting insulin, 2 hour glucose concentrations, and TAG levels were inversely related to childhood BMI while only fasting insulin was inversely related to childhood BMI in men. However, these inverse relationships between CVD risk and BMI were attenuated when percent body fat was used in place of childhood BMI.

In conclusion, the study by Wright et al. answers the question whether overweight children are much more likely to become overweight adults, but not necessary become fat adults. Another optimistic finding from this study is that childhood BMI does not raise CVD risk factors, in fact, it suggests an inverse effect.

Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986-1998. *JAMA* 2001;286:2845-2848.

Wright CM, Parker L, Lamont D, et al. Implications of childhood obesity for adult health: findings from thousand families cohort study. *BMJ* 2001;323:1280-1284.

Key messages

- One in five African-American and Hispanic-American children were obese (defined as BMI of greater than 95% for age and sex). One in eight white children were obese.
- Southern states had a higher incidence of childhood obesity.
- Overweight children were heavier in 1998 than in 1986.
- Overweight children were more likely to become overweight adults, but their likelihood of having more body fat was not determined by their weight as a child.
- CVD risk factors were related to BMI at age 50, but not BMI at age 13.

Big Portions, Big People

As the study reviewed above points out, the prevalence of obesity has reached an epidemic proportion here in the US. And according to findings by Young and Nestle, one major contributor for this problem is the colossal portions of foods we eat away from home. This is a growing problem as more meals are eaten away from home. For example, compared to the 1970s when 34% of all food budgets were spent on foods eaten way from home, in the 1990s it was estimated to be almost half of all food budgets at 47%.

Based on their study, portion sizes began to increase in the 1970s but the rate accelerated during 80s and 90s. It was not uncommon for the ready to eat foods the researchers measured to be significantly bigger than the Food Guide Pyramid and food label portion sizes of the US Department of Agriculture (USDA) and Food and Drug Administration (FDA), respectively. Compared to the USDA's standard portion size, some cookie products measured in the study were 700% larger and cooked pastas, muffins, steaks, and bagels were 480%, 333%, 224%, and 195% over the recommended portions, respectively. From the study, sliced white bread was the only item that met the federal standard for portion size. The researchers found that the growing portion sizes were not limited to prepared food. Compared to the recipes in the old editions of *Joy of Cooking Cookbook*, identical recipes in the new edition yielded less serving indicating larger portions.

According to the researchers, the main reason for the "supersizing" of our foods by the food industry was to "retain and expand market share" as profits for most food items rose consistently when manufacturers increase product size. Large portion size resulted in consumers thinking they got a bargain. Unfortunately, people forget the fact that extra food means extra calories that can result in unwanted weight gain. For example, in one chain, 16-oz soda may cost under 5 cents/oz compared to 2.7 cents/oz for the supersized soda, but at a difference of 200 calories. This caloric difference is dramatic when the whole meal is "supersized." The worst part of all this is that society has accepted these large portions as the standard as demonstrated by larger cup holders in newer cars.

Results from this study clearly show that large food portion size is one cause for the obesity problem that we are facing. However, since obesity is a multi-factorial problem with many etiologies, many other weight loss-weight maintenance techniques need to be implemented along with limiting food portion sizes.

Young LR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health* 2002;92:246-249.

Positive Relationship Between Dietary and Serum Lutein and Zeaxanthin and Macular Pigment Density

earlier studies have shown that dietary and serum lutein and zeaxanthin levels are inversely related to age-related macular degeneration risk. These carotenoids are deposited in the foveal pit of the human retina and protect against macular degeneration by absorbing "short-wave light before it can damage vulnerable lipid-rich membranes in the outer segment of photoreceptors."

In this study, Curran-Celentoaro et al. examined the relationship between dietary intake of lutein and zeaxanthin and serum and retinal concentrations in 280 healthy adults from the Indianapolis area (138 men and 142 women). To minimize the possibility of undiagnosed eye disease, all recruited volunteers were between the ages of 18-50. In addition to completing an extensive questionnaire regarding the lifestyle patterns and health status, dietary intakes were assessed using a food frequency questionnaire and fasting blood samples were collected and analyzed using HPLC to determine carotenoid levels. The food-frequency questionnaire had 122 foods and food groups to estimate the dietary consumption of lutein, zeaxanthin, and other carotenoids. The researchers measured macular pigment optical density (MPOD) with a 460-nm, 1^{o} test stimulus, psychophysical technique.

Analysis of the collected data indicated that 85% of the subjects were white, 25% smokers, 21% former smokers, 32.5% had BMI of greater than 26. The food frequency questionnaire showed that women in the study consumed slightly less calories from fat and 19% more lutein and zeaxanthin than men. Despite the extra lutein and zeaxanthin intake, the average MPOD were similar between the sexes. The average MPOD was 0.207±0.13 in women and 0.215±0.13 in men. The researcher also observed no significant difference in MPOD values among smokers, non-smokers, and former smokers. But according to the researchers, current smokers consumed significantly more fruits, vegetables, and carotenoids than the other 2 groups. (Further analysis showed that there was an inverse dose response relationship between smoking and serum carotenoid levels.) Finally, even though there were great individual differences in MPOD, these data clearly showed that there is a direct relationship between MPOD and serum and dietary lutein and zeaxanthin levels. For example, the regression coefficient between MPOD and serum lutein was 0.26 ($p<0.0001$), and 0.20 ($p<0.0001$) and 0.21 ($p<0.0005$) for the relationship between MPOD and serum zeaxanthin and dietary lutein and zeaxanthin, respectively.

Compared to earlier studies, the average MPOD values in the Midwest cohort were inline with MPOD levels found in a Southwest population with 0.22±0.13, but approximately 40% less than people from the Northeast region. Also, Curran-Celentano et al.'s findings regarding average dietary and serum lutein and zeaxanthin were comparable with other large studies such as the Beaver Dam, Eye Disease Case Control Study, and NHANES III studies.

Based on their findings, the researchers concluded that there is a direct relationship between dietary and serum lutein and zeaxanthin levels and MPOD. Also, in high risk groups for macular degeneration; women, smokers, and persons with lighter-colored irises, higher consumption of lutein and zeaxanthin resulted in increasing MPOD to the level seen in men and non-smokers.

Curran-Celentano J, Hammond BR, Ciulla TA, et al. Relation between dietary intake, serum concentrations, and retinal concentrations of lutein and zeaxanthin in adults in a Midwest population. *Am J Clin Nutr* 2001;74:796-802.

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

Metabolic Syndrome a Growing Problem in US Adults

While experimental evidence associating the metabolic syndrome with increasing diabetes and CVD risk was clear enough for the National Heart Lung Blood Institute's National Cholesterol Education Program committee to stress the importance of treating this condition in their Adult Treatment Panel III (ATP III) report, data on the prevalence of the metabolic syndrome in the US was not available. Therefore, using 8,814 volunteers from the NHANES III study, Ford and colleagues tried to determine a clear association.

Based on the definition of the metabolic syndrome in the ATP III report, a patient must exhibit at least 3 of the following abnormalities: abdominal circumference greater than 102 cm in men and 88 cm in women; serum TAG level of at least 150 mg/dl; HDL cholesterol level of less than 40 mg/dl in men and less than 50 mg/dl in women; blood pressure of at least 130/85 mg Hg; or a serum glucose level of at least 110 mg/dl. Using these criteria, it was determined that 23.7% had the metabolic syndrome disorder. The metabolic syndrome was especially common among people over 60, with a prevalence rate of 43.5% in participants between 60-69 years old and 42% in people over 69, compared to 6.7% in people between 20-29 years old. Like the prevalence of overweight and obesity which coincides with the metabolic syndrome, Ford and colleagues observed a difference in prevalence of the metabolic syndrome among different ethnic groups. Mexican-Americans had the highest incidence of the metabolic syndrome (31.9%) followed by whites (23.8%), blacks (21.6%), and "other" races (20.3%). Also, there was no gender difference in prevalence of the metabolic syndrome in whites or "other" races, but a significant difference in African-Americans and Mexican-Americans. For example, compared to the African-American men and Mexican-American men, prevalence of the metabolic syndrome was 57% and 26% higher among the African-American women and Mexican-American women, respectively. When these numbers were extrapolated using 2000 census data, it was estimated that 47 million American have the metabolic syndrome.

In conclusion, the metabolic syndrome is a major health problem that requires substantial commitment by both health professionals and patients to solve. According to the researchers, health professionals need additional education and training to effectively treat the metabolic syndrome, and patients need to modify behaviors that are thought to be the root cause of the metabolic syndrome (poor nutrition and inactivity).

Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults. Findings from the third national health and nutrition examination survey. *JAMA* 2002;287:356-359.

New Education Materials

We are happy to announce that we have 3 new education materials. These materials can be ordered by contacting ENC at 202-833-8850 or by visiting our website at enc-online.org

- Nutrition Realities: Nutritionally Balanced and Packaged by Nature. This summary report for health professionals discusses the delicate balance between food intake and human metabolism. Highlighted issues include functional foods, dietary supplement use, and vegetarianism.
- Functional Foods Fact Sheet - A short summary of important facts pertaining to functional foods including: current definition, history, labeling and claims specific to functional foods and a discussion of their cost as related to their benefit.
- Special Report on eggs: 2 page review article on the latest studies regarding dietary cholesterol and CVD risk and other beneficial effects of including eggs in a healthy diet.

Very-Low-Birth-Weight Babies Face Challenges Even As Young Adults

Thanks to the introduction of neonatal intensive care units in the 1960s, lives of many very-low-birth weight infants, less than 1,500 grams, have been saved. Yet, according to a long-term follow-up study, even in early adulthood, many of these infants continue to be physically and mentally disadvantaged relative to normal birth weight controls. Hack et al. compared the level of education, cognitive and academic achievement, and rate of chronic illness between VLBW infants born between 1977 and 1979 with normal weight infants during the same period. Based on their findings, compared to 233 controls, 242 young men and women born as VLBW babies faced more challenges. For example, 33% of VLBW participants were affected by chronic medical conditions such as cerebral palsy, blindness, and subnormal height, while only 21% of control group had similar problems. In addition to physical problems, young men and women in the case group had more mental challenges than the control group. Mean IQ scores were 87 in the case and 92 in the control group. The case group was less likely to have graduated from high school or obtained a general equivalency diploma (74%) compared 83% in the control group. Only 16% of the case was enrolled in 4 year college programs compared to 44% from the control group. The data also showed that a higher percent of the case group repeated a grade in school and the mean age at the time of high school graduation was significantly higher. These differences remained significant even after participants with neuro-sensory impairments were excluded from the analysis. However, contrary to the study hypothesis, the case group was less likely to use alcohol and marijuana and have encounters with police for traffic violation or crime. When it came to sexual activity, VLBW males reported being more active and sired more children than normal birth weight males. On the other hand, the response was reversed in females. The VLBW females were less sexually active than the normal birth weight women.

The results from this study confirm Hack et al.'s earlier findings which showed that VLBW children at age 8 were both physically and mentally slower than the normal weight children and that these problems persist even into young adulthood. Lower educational attainment in VLBW children concerned the researchers and its possible role in continuing to disadvantage these children's life.

Hack M, Flannery DJ, Schluchter M., et al. Outcomes in young adulthood for very-low-birth-weight infants. *N Engl J Med* 2002;346:149-157.

Social Status Effects CHD Risk in Men

Previous studies by Barker and colleagues showed that low birth weight and poor infant growth are risk factors for CHD later in life. Low birth weight increased CHD risk by 31% and low weight gain during the first year of life increased CHD risk by 20%. Now they have taken these observations further to show that in men of low birth weight, adult socioeconomic status is a good predictor of CHD risk.

The 3,676 men in this study were born between 1934-1944 in Helsinki University Central Hospital, attended both child welfare clinics and public schools in Helsinki, currently resided in Finland, and participated in the 1980 census. Using records from these institutions, the researchers established a relationship between education, social status, and CHD incidence in men who were underweight as infants. Besides already higher CHD risk, education, social status, and income levels exerted a greater impact on CHD risk in low birth weight men and men with low weight gain during the first year of life. Men in the lowest level of education, lowest income group and working as laborers had the highest hazard ratios for CHD (2.25, 1.71, and 2.15, respectively). Even though the father's social status was not related to birth weight, it did play a direct role in the off-springs' CHD risk. But regardless of parental influences, the relationship between social and education levels and CHD risk was significant.

The simultaneous effects of early body size and socioeconomic status showed that social class and income were 2 factors that raised CHD risk in men from the low ponderal index group (kg/m^3), but had minimal effect on CHD risk in men classified in the group with a higher ponderal index. The researchers observed that in men with a low ponderal index plus accelerated weight gain during early childhood (1-12 years), a decline in social status was associated with higher CHD risk. No such relationship was observed in the low ponderal index group who did not have rapid weight gain during childhood. Finally, the hazard ratios for CHD was 1.19 for each standard deviation decrease in weight at 1 year, 1.22 for each decrease in level of education and 1.17 for each decrease in adult social class.

The results indicate that men who were thin at birth were more susceptible to social influences on CHD risk. Lower social status and educational attainment greatly increased CHD risk in men who were low birth weight infants. This was especially a problem in men who were thin and had high rates of weight gain during childhood. Barker and colleagues suggested that this maybe due to altered liver growth and subsequent re-programming of lipid metabolism and blood coagulation.

Barker DJ, Forsen T, Uutela A et al. Size at birth and resilience to effects of poor living conditions in adult life: longitudinal study. *BMJ* 2001;323:1-5.

Antioxidant Vitamins Show No Protection Against CVD

In their efforts to protect CVD patients against future CVD events, cardiologists commonly treat hyperlipidemics with both statins and supplements of antioxidant vitamins. But surprising findings by Brown et al., are making doctors re-evaluate the use of antioxidant vitamins in CVD patients. In this 3 year, double-blind trial, Brown and colleagues treated 160 coronary disease patients with normal LDL levels, but low HDL with either simvastatin-niacin (n=33), simvastatin-niacin plus antioxidants (n=40), antioxidant vitamins (n=39), or placebo (n=34) and measured the change in plasma lipid and lipoprotein levels and percent change in coronary stenosis during the follow-up period. The average subjects in the study were overweight men in their 50s.

The mean dose of simvastatin and niacin were 13±6 mg/d and 2.4±2.0 gm/d, respectively, but varied depending on the patient's baseline plasma lipid levels. The antioxidant therapy consisted of 800 IU of vitamin A, 1,000 mg vitamin C, 25 mg beta carotene, and 100 µg of selenium. Plasma lipid and lipoprotein levels were regularly checked during clinic visits and arteriography was used to measure the change in blockage of coronary arteries.

According to the data, the compliance rates on all 4 treatment groups were between 80-95%. Also, as expected, plasma lipid, lipoprotein, and apolipoprotein levels improved in the simvastatin-niacin group and the simvastatin-niacin plus antioxidant group. For example, compared to a baseline plasma total cholesterol of 201 mg/dl, LDL cholesterol of 125 mg/dl, TAG of 213 mg/dl, and HDL cholesterol of 31 mg/dl, simvastatin-niacin therapy resulted in 31%, 42%, and 36% decrease in total cholesterol, LDL cholesterol, and TAG level, respectively, while HDL cholesterol levels increased by 26%. HDL_2 and Lp(A-I) also increased by 65% and 81%, respectively. Adding antioxidants to simvastatin-niacin therapy blunted the lipid lowering effect of simvastatin-niacin therapy. For example, adding antioxidant to simvastatin-niacin decreased total cholesterol, LDL cholesterol, and TAG to 27%, 36%, and 31%, respectively. The antioxidant vitamin treatment had no positive effect on plasma lipids, but instead, TAG levels increased and HDL_2 decreased thus resulting in a negative lipid profile. The plasma antioxidant vitamin concentrations did increase following antioxidant treatment, indicating that subjects were taking the supplements. Also, "diene lag time, an index of the resistance of LDL to oxidation, increased by 35%, from 52.4 minutes to 70.5 minutes ($p<0.001$)." Lipid profile did not significantly change in the placebo group.

The arteriography readings showed that in the placebo group, stenosis in proximal arteries progressed by 3.9% from baseline values of 34.5% occlusion, which was the largest change among the 4 groups. Next highest progression of stenosis during follow-up was in the antioxidant group with 1.8%, followed by 0.4% in the simvastatin-niacin plus antioxidant group, and regression of 0.7% in the simvastatin-niacin group. Analysis of the clinical cardiovascular events also showed that, compared to the placebo group, the simvastatin-niacin group had a 90% lower incidence of revascularization, confirmed MI or stroke, and deaths related to CVD.

In conclusion, these results strongly favor the use of lipid lowering statins such as simvastatin in combination with HDL cholesterol raising niacin in hyperlipidemic patients with low HDL levels. Based on the "epidemiological projections of a 1 percent reduction in cardiovascular risk for each 1 percent increase in the HDL cholesterol level and a 1 percent reduction in risk for each 1 percent decrease in LDL cholesterol level," according to the researchers, simvastatin-niacin use in this study would lower CVD risk by 68%. However, the decrease in lipid lowering effect following the addition of antioxidant to simvastatin-niacin treatment was a surprising outcome and makes one question the benefit of using antioxidants in combination with statins. Aside from raising plasma vitamin levels and increasing LDL oxidation time, antioxidant therapy in this study also, did not significantly improve plasma lipid profile or protect against progression of coronary stenosis and cardiovascular events.

Brown BG, Zhao XQ, Chait A, et al. Simvastatin and niacin, antioxidant vitamins, or the combination for the prevention of coronary disease. *N Engl J Med* 2001;345:1583-1592.

Editorial

Imagine a Real World

Imagine working hard your entire life to build a business, establish security for your family, and contribute to the community you live in. Now, imagine that one morning you wake up and it is all in shambles, destroyed because someone doesn't agree with what you do. What you do is perfectly legal, you're honest, customers patronize your business, you pay your taxes, provide jobs in your community, and you raise your family with sound educational and work ethics. But those who don't like what you do destroy it all. Even worse, not many people seem to notice much less care that it happened. Just imagine.

A car dealership that it has taken you years to build has 36 SUVs burned and over a million dollars in damages to the facilities. The warehouse on your family's egg farm is burned to the ground for a $1.5 million loss. Your sand and gravel business is gone after three trucks are burned for a quarter million dollars in damages. And at your home you find your house spray painted, your car overturned, and your name, address and phone numbers provided to a cadre of fanatical nut cases. All in a month's work for those charming social misfits who brought you McMurder and Murder King and Shameway for public display while their shadow cohorts systematically destroy people's lives and livelihoods. ALF (Animal Liberation Front) actually brags that in 2001 they and their allies in the "we know what's best for you" underground accomplished the following: 43 major actions involving setting 20 fires (with some damages running over $5 million), damaging or destroying 19 buildings, damaging or destroying 128 vehicles, smashing or damaging over 200 windows, and with this chaos achieving over $17.3 million in damages to those they disagree with.

Let's break this down a little further: almost 1 action a week resulting in $47,400 per day in damages, not counting such "cute" tricks as gluing locks of cars and buildings, spray paintings, tree spikings, vehicle and machine monkey-wrenchings, and the chronic harassment of specific individuals, their spouses and their children. ALF took special pride in a successful "action" by cousin in crime ELF (Earth Liberation Front) which succeeded in burning down the Center for Urban Horticulture at the University of Washington in Seattle which expanded their damage estimate by $5.6 million. Oh how proud to achieve such marvels. And what sin did a bunch of university types in horticulture do to deserve this fate? They were doing research on genetically modified crops, what else!

It has been written that the United States of America is a critical experiment to determine whether people are able to rule themselves through reasoned argument. The experiment seems to be in trouble! I don't like what you do therefore I have the right to stop you from doing it. I do not care whether what you do is legal and whether the majority of people are your customers, or whether you have your own set of rights as an American. I do not like what you do! And I have the right, no, I believe that I have the obligation if my ethics and morality are exact, to stop you, by any means necessary. Reasoned arguments? Sounds more like terrorism. At least terrorism as defined in dictionaries, *"the unlawful use or threatened use of force or violence by a person or an organized group against people or property with the intention of intimidating or coercing societies or governments, often for ideological or political reasons."* Hardly passes the test for reasoned arguments. But then what are the alternatives if your arguments are in fact not reasoned?

So after years of hard work and study you've gotten your degree and certification, you've invested your life savings to get your new offices ready, you've built your clientele through professionalism and dedication, and you are ready to offer your services for the health and improvement of the community. But, you advise them to eat a diet with balance, variety and moderation including animal products. You counsel that all foods provide essential nutrients, even meat, eggs, and fish. You do not demand that all your clients be vegetarians, that they raise their own organic crops, that they stop their obvious materialistic consumerism for the benefit of the planet. I don't like what you do and how you do it and the way you teach it. Furthermore, you own stocks in unapproved companies, you patronize restaurants chains living off of animals, you wear clothing made in sweat shops, you drive a polluting car, and your new house destroyed the environment. It is just and proper and appropriate that my friends and I burn down your new offices, intimidate and scare away your clientele, harass you at work and at home, spray paint your car, tag your house, glue your locks, and generally make your life hell until you understand that you have no choice but to do it all my way. Just imagine.

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

Eating Frequency Lowers Total and LDL Cholesterol

According to a large cross-sectional, population study out of Norfolk, England, if people are trying to control their plasma lipid levels, it is not only important to watch what they eat, but also how frequently they eat. Based on the European Prospective Investigation into Cancer (EPIC-Norfolk) study, with 14,666 adults, plasma total and LDL cholesterol levels were inversely related to the number of meals volunteers consumed per day. (Frequency of eating included both meals and snacks.) For example, compared to the group that ate only1-2 time per day (n=353), the group that ate more than 6 times per day (n=625) had mean total cholesterol and LDL cholesterol levels of 232 mg/dl and 149 mg/dl, respectively, which represents a lower total and LDL cholesterol by 6.8 mg/dl. High eating frequency also decreased plasma HDL cholesterol levels but to a lesser degree than LDL cholesterol. As a result, the ratio of LDL cholesterol to HDL cholesterol also decreased with more frequent food intake. This finding is somewhat surprising, since high eating frequency was associated with high total calories, fat, carbohydrate, and protein intake. Polyunsaturated fatty acid and saturated fatty acid intake also increased. On the other hand, people who ate less often were more likely to drink alcohol, smoke, and hold sedentary jobs.

The inverse relationship between total and LDL cholesterol and eating frequency continued to be significant even after the researchers adjusted for both lifestyle and dietary factors associated with CVD. Multivariate analysis also showed an inverse relationship between eating frequency and HDL cholesterol, waist-to-hip ratio, and BMI. Age also made a difference in this relationship. The multivariate regression coefficient between total cholesterol and eating frequency was greater in the 45-59 year old cohort (-0.055) compared to the regression coefficient of -0.041 in the 60-75 year old group.

The results from this large cross-sectional study confirm many smaller studies which found an inverse relationship between eating frequency and plasma total and LDL cholesterol concentrations. The beneficial effects of lowering lipids with more frequent feeding has been termed "adaptive hyperlipogenesis." It is thought that by eating more frequently, our body continues to metabolize nutrients by increasing glucose absorption, pancreatic enzyme activity, and hepatic synthesis of fatty acids instead of storing energy. The researchers concluded that frequency of food consumption is an important factor to consider in lipid lowering diet plans.

Titan SMO, Bingham S, Welch A, et al. Frequency of eating and concentrations of serum cholesterol in the Norfolk population of the European prospective investigation into cancer (EPIC-Norfolk): cross sectional study. *BMJ* 2001;323:1-5.

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NUTRITION closeUP

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