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INSIDE

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

Lifestyle Factors Decrease Risk of the Metabolic Syndrome

Metabolic syndrome—a cluster of conditions including glucose intolerance, insulin resistance, large waist circumference, dyslipidemia, and hypertension—is estimated to be present in approximately 1 in every 4 adults in the U.S. Because it is associated with increased risk of cardiovascular disease (CVD) and type 2 diabetes, preventing this syndrome is a high priority among health professionals. Multiple lifestyle factors such as smoking status, drinking habits, diet (especially carbohydrate, fat, and total calorie intake), and physical activity have been associated with the condition; however, the relative influence of these behaviors on the likelihood of developing metabolic syndrome is unknown.

Zhu et al. gathered data from the Third National Health and Nutrition Examination Survey (NHANES III) to evaluate the influence of specific lifestyle factors on the development of metabolic syndrome. Data collection for NHANES III began in 1988 and ended in 1994. Data from 11,239 (5415 men and 5824 women over age 20) out of 40,000 individuals who originally participated in the survey were used in this study. Non-modifiable lifestyle factors included in the analysis were age, ethnicity, education, and household income level. Modifiable lifestyle factors were physical activity, fat and carbohydrate intake, alcohol consumption, and smoking habits. Individuals with three or more of the following risk factors were classified as having metabolic syndrome: waist circumference >102 cm (men) or >88 cm (women) indicating abdominal obesity, triglyceride levels >150 mg/dL, HDL cholesterol levels <40 mg/dL for men and <50 mg/dL for women, high blood pressure, and elevated fasting plasma glucose.

Participants were classified according to carbohydrate intake (low <40%, moderate 40-60%, and high >60% of total calorie intake) and fat intake (low <30%, moderate 30-40%, and high >40% of total calorie intake). Five low-risk categories were chosen for purposes of comparison: normal weight (BMI <25 kg/m^2) or non-obese (BMI <30 kg/m^2), physically active, low to moderate carbohydrate intake, low to moderate alcohol intake, and lifetime non-smoker.

The men were more likely than the women in this study to be classified as having metabolic syndrome (23.0% vs. 21.9%; $P<0.05$). The prevalence of metabolic syndrome among male participants categorized as normal, overweight, and obese was 5.3%, 21.9%, and 58.8%, respectively. The prevalence of metabolic syndrome in normal, overweight, and obese women

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was 5.5%, 27.5%, and 48.8%, respectively. The odds ratios for having metabolic syndrome decreased with increasing reported levels of physical activity.

Men whose reported carbohydrate intake was low to moderate were >50% less likely to develop metabolic syndrome than those whose carbohydrate consumption was high. Carbohydrate intake had little or no effect on the odds ratios for women. High intake of fat was associated with a lower risk of having metabolic syndrome in both men and women. This association was lost after correction for modifiable and non-modifiable lifestyle factors. The protective effect of low to moderate carbohydrate consumption in men is not surprising since high carbohydrate intake is associated with decreased HDL cholesterol concentrations and increased triglycerides. Why the same effect was not observed in women is not fully understood; however, it has been previously reported that high carbohydrate intake affects triglyceride concentrations to a greater extent in men than in women. Because this study did not classify carbohydrates as simple sugars or complex carbohydrates, the differential effects of these two families of carbohydrates remain unknown.

In women, light-to-moderate and heavy alcohol consumption was associated with a lower risk of developing metabolic syndrome, even after controlling for potentially confounding lifestyle factors. Heavy drinking in overweight men was associated with an increased metabolic syndrome risk (OR = 1.37; CI, 1.04 to 1.79; P<0.05). For both men and women, compared to being a current smoker, never having smoked was associated with a lower risk of developing metabolic syndrome.

Regardless of BMI category, physical activity was associated with 31% and 17% risk reductions in men and women, respectively. Participants with BMI less than 30 kg/m^2 who were physically active were 71% and 79% (men vs. women) less likely to have metabolic syndrome, while those with BMI less than 25 kg/m^2 were 84% and 94% (men vs. women) less likely to have metabolic syndrome. Physical activity is thought to play many roles in risk reduction. It has been shown to increase HDL cholesterol, decrease triglycerides, decrease blood pressure, and improve insulin sensitivity and glucose tolerance, thus improving several risk factors for metabolic syndrome.

Non-smoking men and women who were physically active and who reported low to moderate carbohydrate intake (for men) or light-to-moderate alcohol consumption (for women) had odds ratios of 0.52 and 0.54, respectively. When these behaviors were combined with a BMI of <30 or <25, the risk of developing metabolic syndrome was even lower, (73% and 85% lower for men and 90% and 94% lower for women, respectively).

Because these data were gathered in a cross-sectional manner, the relationship between metabolic syndrome and associated lifestyle factors cannot be assumed to be causal. However, these results are valuable in assessing the relative strength of influential lifestyle factors and in helping give direction to future longitudinal studies. These results suggest that physical activity, lower carbohydrate consumption (for men), light-to-moderate drinking habits (for women), not smoking, and maintaining a BMI less than 30 kg/m^2 could help the majority of Americans avoid developing metabolic syndrome.

Zhu S, St-Onge M, Heshka S, Heymsfield S. Lifestyle behaviors associated with lower risk of having the metabolic syndrome. *Metabolism* 2004; 53(11):1503-1511.

Key messages

- Overall, non-smoking individuals who consume diets relatively low in carbohydrates, engage in regular physical activity, drink alcohol in moderation, and keep their BMI within the non-obese range are significantly less likely to develop metabolic syndrome.
- In women, but not in men, moderate alcohol consumption was associated with a lower risk of developing metabolic syndrome.
- In men, but not in women, low to moderate (compared to high) carbohydrate intake appeared to reduce the risk of developing metabolic syndrome.

Glycemic Load Predicts HDL Cholesterol Levels in Youth

Once simply defined as a macronutrient providing 4 calories per gram, the carbohydrate has increased in complexity in both research and consumer realms. Questions must now be answered such as, "Is the food composed of simple sugars or complex carbohydrates?" "Is it whole-grain or made from refined flour?" "Does it increase the postprandial insulin response a lot or a little?" These questions are increasingly significant as evolving research repeatedly demonstrates that different types of carbohydrates vary with respect to their impact on insulin sensitivity, blood lipid profiles, and other factors related to the metabolic syndrome.

It is recognized that a growing number of overweight and obese adolescents and young adults are exhibiting signs of the metabolic syndrome such as high blood pressure, insulin resistance, and dyslipidemia (low HDL and elevated triglycerides). Slyper et al. recently conducted a randomized clinical trial to assess the impact of glycemic load (a predictive measure of postprandial glycemic response) on blood lipid levels in adolescents and young adults.

Participants were healthy males and females (n=32) aged 11-25 years with varying lipid profiles (normolipidemic to hyperlipidemic) and age-adjusted BMI scores. Following a 12-hour fast, blood samples were collected for measurement of blood lipids. Height, weight, and blood pressure were recorded for each participant. They were then instructed to record all daily food intake for 2 weekdays and 1 weekend day to provide a "snapshot" representative of their usual eating habits and to determine the typical daily glycemic load for each participant. Glycemic load was determined by multiplying the glycemic index of each food by its carbohydrate content in grams. Daily glycemic load was determined by summing the glycemic load for each food consumed in a given day.

Correlations and multiple regression analyses were undertaken to determine which dietary components were associated with blood lipid levels. HDL cholesterol was the only blood lipid significantly influenced by any of the dietary components measured in this study. Glycemic load (-0.46; $P<0.01$), glycemic index (-0.37; $P<0.05$), total sugar (-0.38; $P<0.05$), total carbohydrate (-0.41; $P<0.05$), and fructose (-0.41; $P<0.05$) were all inversely correlated with HDL cholesterol levels. Glycemic load was an independent predictor of HDL cholesterol levels, accounting for 21.1% of the variance.

Interestingly, while glycemic load was inversely correlated with percent calories from protein and fat, there was a positive correlation between glycemic load and total protein and fat intake. This would suggest that consumption of foods with a lower glycemic load was associated with higher protein:carbohydrate and fat:carbohydrate intake ratios and that intake of foods with a higher glycemic load was associated with greater overall intake (more carbohydrates, more fat, more protein—in other words, more calories). This indicates that consuming lower glycemic index foods contributes to overall satiety.

Therapeutic lipid-lowering diets are typically low in fat. Patients following such diets have traditionally been instructed to avoid high-fat foods , but might not have been instructed in the best ways to replace high-fat items with healthier fare. Often, the calories from fat are simply replaced by refined carbohydrates. Indeed, research suggests that low-fat diets have been associated with increased consumption of simple sugars, translating to an increased glycemic load. Observations by Slyper et al. draw attention to the potential for significant reductions in HDL cholesterol with increased glycemic load in young patients following low-fat diets.

Slyper A, Jurva J, Pleuss J, et al. Influence of glycemic load on HDL cholesterol in youth. *Am J Clin Nutr* 2005;81:376-379.

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

Fish Intake Slows Progression of Atherosclerosis in Women

Fish intake has traditionally been associated with a reduced risk of CHD mortality, particularly with decreased incidence of sudden death due to coronary events. This association is thought to be related to anti-inflammatory properties of n-3 fatty acids and/or protection against cardiac arrhythmia. However, the influence of n-3 fatty acids from fish on the underlying disease progression is unclear. To address this question, Erkkilä et al. examined the influence of fish intake on the progression of coronary atherosclerosis in postmenopausal women.

Postmenopausal women with established coronary stenosis (>30% reduction in luminal diameter) were eligible to participate in this study. At baseline, all participants provided information on health status, health history, and dietary intake (semiquantitative food-frequency questionnaire). Cardiovascular disease risk factors were assessed and women underwent quantitative coronary angiography to evaluate the presence of coronary stenoses, a process that was repeated ~3 years later to assess atherosclerotic progression.

Participants (n=248) were classified based on fish intake and (because of previous studies linking fish intake to decreased CAD risk in diabetic women) presence or absence of diabetes. Forty-two percent of participants had diabetes at baseline. Demographic data showed that women who reported eating ≥2 servings of fish/week had a higher degree of education. They exercised strenuously more often, had lower carbohydrate intakes, and consumed more calories, protein, alcohol, carotene, and cholesterol. Baseline mean percentage coronary stenosis was greater among diabetic women who were consuming ≥2 servings of fish/week (versus those consuming <2 servings/week).

Women who reported eating ≥2 servings of fish per week experienced smaller reductions in mean coronary artery diameter over 3 years of follow-up than those who reported eating <2 servings/week (P for fish intake = 0.02). This association was lost after adjustment for age, study clinic, race, BMI, smoking, use of cholesterol-lowering medications, hormone replacement therapy, energy intake, diabetes, and other non-dietary factors (model 1; P = 0.06), but was restored after adjustment for intake of nutrients thought to influence CAD risk such as saturated fat, MUFA, PUFA, cholesterol, fiber, and alcohol (model 2; P = 0.006). Those consuming more servings of fish also experienced smaller increases in mean percentage stenosis as compared with those reporting lower fish intake. This association remained significant only for diabetic women after adjusting for variables in models 1 and 2.

Tuna and dark fish are typically rich in n-3, the component of fish oil thought to provide cardioprotective benefits. When fish intake was classified by type of fish (tuna or dark fish vs. "other" types of fish), participants reporting consumption of one or more servings of tuna or dark fish per week experienced smaller reductions in coronary artery diameter (P = 0.02), however, this observation was attenuated and lost statistical significance following adjustment for potentially confounding factors included in model 1 such as age, study clinic, time of follow-up, race, BMI, smoking, use of cholesterol-lowering drugs, hormone replacement therapy, energy intake, and diabetes (although the association remained significant for diabetic women). Following further adjustment for intake of nutrients known to influence CAD risk, the association was again significant (P = 0.02). Among diabetic women, those consuming at least one serving of "other fish" per week experienced smaller reductions in coronary artery diameter and smaller increases in percentage stenosis. Among diabetic women, the consumption of >1 serving of "other fish" was associated with smaller coronary artery diameter and greater percentage stenosis at baseline compared to those consuming less than 1 serving of "other fish" per week.

Women who reported consuming ≥2 servings of fish per week developed fewer new stenoses over the course of the study (P = 0.02). Regarding inflammatory markers (CRP, VCAM-1, IL-6, and ICAM-1), only VCAM-1 was significantly associated with fish intake after adjusting for BMI. As reported fish intake rose, VCAM-1 concentrations decreased. None of the other inflammatory markers were significantly associated.

Based on the data presented in this study, consumption of fish (especially dark fish) appears to be associated with reduced progression of atherosclerosis in postmenopausal women with existing CAD. In agreement with previous research, frequent consumption of fish appears to be especially protective in women with diabetes.

Erkkilä A, Lichtenstein A, Mozaffarian D, Herrington D. Fish intake is associated with a reduced progression of coronary artery atherosclerosis in postmenopausal women with coronary artery disease. *Am J Clin Nutr* 2004;80:626-32.

Metabolic Consequences of Regular vs. Irregular Meal Patterns

Meal frequency has long been a topic of curiosity among those hoping to lose weight. Does eating more often help stave off excess pounds? Although an early study in the 1960s found an inverse association between meal frequency and body weight, subsequent research examining the influence of meal frequency on energy intake and postprandial energy expenditure has been largely inconclusive. Glucose and insulin responses have also been evaluated in relation to meal frequency with equivocal results. Irregular meal frequency was recently shown to decrease postprandial energy expenditure in healthy lean women. Insulin resistance increased and fasting lipid levels worsened after a period of sustained irregularity in meal frequency.

The same group of researchers undertook a randomized crossover trial in otherwise healthy obese women to determine whether irregular patterns of meal frequency have similar metabolic consequences in obese individuals.

Ten women were recruited to participate in this two-phase, 42-day crossover study. Although obese, all women were otherwise healthy. Those with hyperlipidemia, hyperglycemia, or who were dieting or experiencing depression were excluded from the study. All participants were asked to maintain their regular food intake over the first 14 days. They would do this by eating 6 evenly-spaced meals per day (regular meal pattern) or by following an irregular meal pattern of anywhere from 3 to 9 eating occasions per day. In either case, participants were expected to maintain their habitual intake (food selections and quantities). Participants went back to their normal patterns of food intake for 14 days prior to the initiation of phase 2, in which the women were assigned to follow the alternative eating pattern (regular or irregular).

The women completed semi-quantitative food intake records prior to phase 1 of the study and on the 3rd, 11th, and 14th days of phases 1 and 2. On the first and last days of phases 1 and 2, height, weight, and waist and hip circumferences were measured and recorded for all participants. Blood samples were taken before and after a milkshake test meal to measure insulin, glucose, and plasma catecholamine responses. Fasting concentrations of total, LDL, and HDL cholesterol were also measured. Resting metabolic rate (RMR) was measured and hunger was assessed before and after the milkshake test meal. Insulin resistance was evaluated using homeostasis model assessment (HOMA-IR).

Neither body weight, nor body-fat composition changed for participants in either group following phase 1 or 2. However, participants reported lower mean energy intakes during the regular meal pattern when compared with the irregular meal pattern (7.98±0.49 MJ/d vs. 8.32±0.35 MJ/d; $P<0.01$). Although no differences were reported in appetite ratings following the test meals during either stage, participants reported higher energy intake when consuming 9 versus 3 meals per day when following the irregular meal pattern. Reported macronutrient intake did not differ between the two intake patterns or between days within each phase. Neither meal pattern significantly influenced fasting RMR in these participants, however, the postprandial metabolic rate (PPMR) declined significantly following the irregular meal pattern and rose significantly following the regular meal pattern. The thermic effect of foods (TEF) decreased from baseline following the irregular meal pattern and increased from baseline following the regular meal pattern (paired t test; $P=0.008$ and $P=0.018$, respectively).

Fasting blood glucose remained the same following both meal patterns, however, the peak serum insulin concentration was significantly lower following the regular meal pattern ($P=0.001$) and was significantly higher following the irregular meal pattern ($P=0.021$). Plasma total cholesterol was lower following the regular vs. irregular meal pattern ($P=0.003$), as were plasma LDL cholesterol concentrations. These differences were not associated with differences in mean energy intake between the two meal patterns. Plasma HDL and triacylglycerol concentrations did not differ between meal patterns. Plasma norepinephrine also remained unchanged.

The researchers conclude that for these obese women, irregular meal patterns resulted in decreased postprandial energy expenditure and TEF, increased total and LDL cholesterol, and impaired postprandial insulin sensitivity (but no change in fasting insulin sensitivity according to the HOMA-IR index). These results support those of previous research by Farschi and colleagues showing similar responses in healthy lean women. Because body weight and body-fat composition remained unchanged over the course of the study, no conclusions can be made about whether such a meal pattern contributes to weight gain; however, a longer study with more participants would likely be required to detect such differences.

Farschi HR, Taylor MA, Macdonald IA. Beneficial metabolic effects of regular meal frequency on dietary thermogenesis, insulin sensitivity, and fasting lipid profiles in healthy obese women. *Am J Clin Nutr* 2005;81:16-24.

Influence of Two Methods of Dietary Fat Reduction on CVD Risk Factors

Dietary intervention strategies for decreasing dietary fat consumption include two basic methods: substitution and reduction. The substitution method takes advantage of lower-fat versions of full-fat products. Low-fat or fat-free versions are eaten in place of higher-fat products typically consumed (ex. replacing whole milk with 1% milk). The "reduction" method includes replacing higher-fat foods with foods that are naturally low in fat or fat-free (ex. replacing whole milk with water). Whether one strategy is more effective than the other in modifying cardiovascular risk factors is not known. To address this question, Heald and colleagues studied the influence of these two dietary interventions on risk factors such as body weight, insulin-like growth factor (IGF)-1, and C-reactive protein.

Eighty volunteers were recruited to participate in this randomized intervention study. All were women over 18 years of age who had been previously screened and categorized as consumers of high-fat diets. Each was randomized to one of 4 treatment groups (substitution, reduction, combination, or control). Women in the substitution group (SUB) were instructed not to alter their usual diet (including portion sizes), but to substitute high-fat foods typically consumed with reduced-fat alternatives. (Red meats were to be replaced with chicken or fish.) Those in the reduction group (RED) were instructed to reduce their intake of high-fat foods and increase their consumption of foods that are naturally low in fat. Women in this group were also asked to reduce portion sizes of foods high in fat. They were instructed to increase high-fiber foods such as bread, pasta, rice, cereals, and potatoes. The combination group (COM) was asked to replace high-fat items with reduced-fat versions and to decrease their consumption of high-fat foods. The COM group was also asked to increase fiber-rich foods and/or decrease the portion size of high-fat foods. Women in the control group (CONTROL) were asked not to make any changes to their normal diet. Participants in all groups were instructed not to change their patterns of physical activity for the duration of the study.

Volunteers completed food frequency questionnaires before and after the intervention to assess changes in fat intake. Four-day intake records (covering 3 weekdays and 1 weekend day) were also completed by each participant at baseline, after 1 month, and at the completion of the study (3 months). Anthropometrics (body weight and percent body fat [measured by bioelectrical impedance]), in addition to laboratory values (lipid profile, glucose, C-reactive protein, insulin, IGF-1, and IGF binding protein-1) were measured at baseline and again post-intervention. Participants were similar at baseline with regard to age, ethnicity, baseline body weight, BMI, smoking status, total and saturated fat intake, and the laboratory values detailed above. Both body weight [-0.4 kg (95% CI -1.3, 0.4)] and percentage body fat [-1.3% (95% CI -2.0. -0.5)] decreased significantly for women in the SUB group compared to baseline. No other group experienced significant changes in weight or percentage body fat over the course of the study. Although all groups experienced a decline in fat intake over the course of the study, the SUB group demonstrated the greatest reduction ($P<0.01$).

Fasting triacylglycerols (-0.2 mmol/l, $P=0.04$) and C-reactive protein (-24.3%, $P=0.04$) dropped significantly from baseline for participants in the SUB group. Researchers also observed a trend toward decreased blood cholesterol levels following the SUB intervention. These results were not observed in any other group. Serum levels of IGF-1 increased significantly following the SUB (31 ng/ml; $P=0.02$) and RED (19 ng/ml; $P=0.02$) interventions. There were no changes in HDL cholesterol levels, IGF binding protein-1, fasting insulin, fasting glucose, or HOMA-S (measure of insulin sensitivity) over the course of the study for any of the interventions.

These findings suggest that using the substitution method (using reduced-fat versions of full-fat products) to decrease overall fat intake is more effective than using the reduction or combination methods as previously described. The data also indicate that the substitution method results in greater reductions in weight and percentage body fat. Based on the data provided by Heald et al., it is not possible to determine the factors responsible for the success of the SUB method. These could have included decreased fat intake, decreased overall energy intake, or enhanced palatability and acceptability (and thus, greater compliance because the required dietary changes were perceived as smaller than those required for the RED intervention). No information was provided about dietary changes other than fat intake, therefore, whether participants in the RED intervention replaced high-fat foods with low-fat protein sources, whole-grain carbohydrates, or refined carbohydrates is not known.

The substitution method was more effective than any other intervention in modifying CVD risk factors. C-reactive protein is a strong predictor of CVD events and IGF-1 is thought to protect against the development of glucose intolerance and CVD. It is significant that C-reactive protein, IGF-1, and triacylglycerol levels were improved (decreased C-reactive protein and triacylglycerol levels and

increased IGF-1) after a relatively short (3 month) intervention. These findings suggest that substituting full-fat foods with their reduced-fat versions (a method already known to be one of the most highly acceptable and easily adopted) is more effective in reducing dietary fat intake, decreasing body weight and percentage body fat, and improving cardiovascular risk profile than reducing fat by eliminating high-fat favorites and replacing them with foods that are naturally low in fat.

Heald AH, Golding C, Sharma R, et al. A substitution model of dietary manipulation is an effective means of optimizing lipid profile, reducing C-reactive protein and increasing insulin-like growth factor-1. *Br J Nutr* 2004;92:809-818.

Editorial: *"It's Dèjá Vu All Over Again"**

You know you have been in the same line of work way too long when old, discarded concepts reappear as brand new ideas. In the 1970s I listened with great interest to the arguments of outstanding scientists like E.H. Ahrens, Jr., M.D. and David Kritchevsky, Ph.D. regarding the potential problems with a "one diet fits all" approach to dietary recommendations. Part of the argument was simple—humans are not like an inbred strain of research animals. Why would anyone expect all humans to respond the same way to the same diet and attain the same health benefits? Many book chapters were written addressing this issue and many respected scientists were accused of trying to ruin the massive public health approach to having everyone live long and healthy lives thanks to the established dietary guidelines. Nirvana could be achieved if only the naysayers would shut up and go away. Fortunately (or unfortunately depending upon your perspective) they hung around and kept making noise and must have left some remnants of the concept because it was the hot topic in nutrition at the Experimental Biology 2005 meetings held in San Diego, California this year.

Nutrigenomics is the catch phrase for individual heterogeneity of physiological responses to nutrients. (Maybe the reason it didn't catch on in the 70s was that there wasn't a new, catchy phrase to hook people on the concept—sort of like having the right acronym for a clinical trial.) We've known about this heterogeneity of responses to diet in the area of blood lipids and lipoproteins for thirty years but it seems that it just now is reaching those who make nutrition policy. Sort of like when the concept of people eating foods rather than nutrients and having dietary patterns rather than experimental meal plans finally found its way into the Dietary Guidelines for Americans. Even the new MyPyramid incorporates gender, weight and activity level to determine a dietary pattern. Yes indeed, there really are nutritional differences between a 24 year old, 5'4'', 120 lb female and a 63 year old, 5'11'', 230 lb male. And we're actually going to start treating them with targeted nutritional information to address their specific needs. But for the vast majority of you who are younger than I am, do not be fooled into thinking this is a new idea. It has its history; and a number of good scientists were marginalized for supporting the concept before its time. (There is great truth in George Santayana's comment that "Those who cannot remember the past are condemned to repeat it.")

With that being said, the question raised years ago remains the same today—How do we test everyone in the population to define their genetic profiles and determine optimal nutrient intakes and sensitivities? Some will be relatively easy based on phenotypic expression whereas others will not even be defined for decades. How long will it be before we can run a genetic profile as part of our clinical screening procedures? Before we can make specific recommendations, based on genetics, for our patients? Even more disconcerting, how long before you can get that genetic profile and dietary prescription for only three payments of $39.99 plus shipping and handling? In the meantime it's probably a good idea to apply the current dietary guidelines within the context of the needs of the individual patient—tempered with common sense. (Just remember, common sense is not so common.) Our dietary guidelines have changed significantly over the last decade, especially in terms of a de-emphasis on the calories from total fat and a re-emphasis on fruits, vegetables, whole grains and physical activity. We should apply these concepts to our educational efforts and get away from berating this product or that food and focus on positive messages promoting eating well for health.

*Yogi Berra

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

Dark Chocolate and Insulin Sensitivity

The good news about chocolate keeps rolling in. It is thought that the flavonols in cocoa and chocolate play a role in protecting the vascular endothelium by mediating nitric oxide availability, which is key in eliciting appropriate blood vessel responses to vasopressure. Indeed, dark chocolate high in polyphenols has been shown to reduce blood pressure in elderly individuals with hypertension. Nitric oxide levels have also been associated with insulin sensitivity. In an effort to shed further light on this relationship, a recent study tested the effects of polyphenol-rich dark chocolate versus white chocolate on blood pressure and insulin sensitivity in healthy adults.

Fifteen adults aged 33.9±7.6 years were recruited to participate in this randomized crossover study. None were found to have impaired insulin sensitivity, type 2 diabetes, or elevated fasting glucose levels. Following a 7-day chocolate-free washout period, participants were assigned to consume either 100 g of dark chocolate (containing ~500 mg polyphenols and 480 kcals) or 90 g white chocolate (containing no known polyphenols and 480 kcals) each day for a period of 15 days. The white and dark chocolate bars were similar in cocoa butter, fiber, electrolyte, and vitamin content, as well as macronutrient composition. Following this initial trial period, participants underwent another 7-day choclate-free washout period, after which they began another 15 days on the alternate chocolate regimen.

Oral glucose tolerance tests were administered to all participants on day 7 of the pre-intervention washout period to obtain baseline measures of insulin resistance (homeostasis model assessment [HOMA-IR]) and insulin sensitivity (quantitative insulin sensitivity check index [QUICKI] and insulin sensitivity index [ISI]). Oral glucose tolerance tests were again administered at the end of each 15-day intervention phase.

Indices of insulin sensitivity were significantly higher following 15 days of dark chocolate when compared to those following 15 days of white chocolate consumption (QUICKI, P=0.001, paired t test). HOMA-IR was lower following the dark chocolate regimen vs. the white chocolate regimen (P<0.001, paired t test). ISI values were also higher following the dark chocolate than after the white chocolate intervention (15.18±7.69 vs. 7.4±3.5; P=0.001).

These data suggest that chocolate rich in polyphenols has the potential not only to reduce blood pressure, but to improve insulin sensitivity. Although further research in this area is warranted to confirm a cause-effect relationship and to clarify the mechanisms by which it occurs, the observations made by Grassi et al. related to insulin sensitivity represent a significant and novel finding in this relatively new area of research.

Grassi D, Lippi C, Necozione S, et al. Short-term administration of dark chocolate is followed by a significant increase in insulin sensitivity and a decrease in blood pressure in healthy persons. *Am J Clin Nutr* 2005;81:611-4.

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