The Changing Roles of Dietary Carbohydrates
From Simple to Complex
Amy E. Griel, Elizabeth H. Ruder, Penny M. Kris-Etherton

Abstract—The dietary recommendations made for carbohydrate intake by many organizations/agencies have changed over time. Early recommendations were based on the need to ensure dietary sufficiency and focused on meeting micronutrient intake requirements. Because carbohydrate-containing foods are a rich source of micronutrients starches, grains, fruits, and vegetables became the foundation of dietary guidance, including the base of the US Department of Agriculture’s Food Guide Pyramid. Dietary sufficiency recommendations were followed by recommendations to reduce cholesterol levels and the risk for cardiovascular disease; reduction in total fat (and hence saturated fat) predominated. Beginning in the 1970s, carbohydrates were recommended as the preferred substitute for fat by the American Heart Association and others to achieve the recommended successive reductions in total fat and low-density lipoprotein cholesterol (LDL-C). Additional research on fats and fatty acids found that monounsaturated fatty acids could serve as an alternative substitution for saturated fats, providing equivalent lowering of LDL-C without concomitant reductions in high-density lipoprotein cholesterol witnessed when carbohydrates replace saturated fat. This research led to a sharper focus in the guidelines in the 1990s toward restricting saturated fat and liberalizing a range of intake of total fat. Higher-fat diets, still low in saturated fatty acids, became alternative strategies to lower-fat diets. As the population has become increasingly overweight and obese, the emergence of the metabolic syndrome and its associated disruptions in glucose and lipid metabolism has led to reconsiderations of the role of carbohydrate-containing foods in the American diet. Consequently, a review of the evidence for and against high-carbohydrate diets is important to put this controversy into perspective. The current dietary recommendations for carbohydrate intake are supported by the evidence. (Arterioscler Thromb Vasc Biol. 2006;26:000-000.)

Key Words:

Data from the National Health and Nutrition Examination Surveys have been collected by the Centers for Disease Control since the 1970s to track dietary habits of Americans. Careful comparison of these surveys, taking into account improved methodologies for collecting intake data,1–3 indicate that over the past 25 years, the consumption of carbohydrate in the United States has increased from 42.4% kcal to 49.0% kcal for men and from 44.8% kcal to 49.7% kcal for women.1 The increase in carbohydrate intake has been accompanied by an apparent increase in energy intake (2450 kcal to 2618 kcal for men and 1542 kcal to 1877 kcal for women; \( P<0.01 \)), and a corresponding decrease in the percentage of calories from total fat (36.9% to 32.8%; \( P<0.01 \) for men and 36.1% to 32.8%; \( P<0.01 \) for women).1 Based on our best estimates, Americans consume more calories and more carbohydrates than ever before.

The increase in carbohydrate content of the diet brings the dietary intake of carbohydrates well within the range of current dietary guidelines. Specifically, the Institute of Medicine of the National Academies recommends a diet that provides 45% to 65% kcal from carbohydrates, 10% to 35% kcal from protein, and 20% to 35% kcal from total fat, while keeping saturated fat, trans fat and dietary cholesterol low.4 These recommendations are consistent with the Dietary Guidelines for Americans 2005.5 The American Heart Association and the National Cholesterol Education Program recommend a diet that provides 50% to 60% kcal from carbohydrates. Irrespective of total calories, Americans are consuming the recommended percent calories from carbohydrates, but how does the quality of these carbohydrates affect lipids and lipoproteins?

Lipid Modifying Effects of Carbohydrate Rich Diets

It has been universally documented that when carbohydrates are substituted for saturated fat, a reduction in total and low-density lipoprotein cholesterol (LDL-C) is observed.6–18 These observations led to the development of a 3-step American Heart Association diet for the treatment of hypercholesterolemia, in which successive reductions in total fat were recommended for control of lipids. High-carbohydrate diets remained the diet of choice until research on monoun-
HDL-C and TG.4 Weighted least-squares regression analysis between total fat content of the diet and the changes in feeding studies, there is a linear dose-response relationship range of total fat (18% to 40% kcal) evaluated in controlled high-density lipoprotein cholesterol (HDL-C). Within the propensity to increase fasting triglycerides (TG) and reduce saturated fat, rather than total fat.

American Heart Association dietary guidelines appropriately diets were compared: high-carbohydrate, high-protein, and Portfolio Diet Study (2003)25 Randomized, controlled atherogenic lipoprotein, also increase in a stepwise manner as be expected to increase 6%. Levels of Lp(a), a lesser studied revealed that for every 5% decrease in total fat, HDL-C levels lent to high-carbohydrate, low-fat diets.20 As the monounsatur-

saturated fatty acids, a more ubiquitous fatty acid than polyunsaturated fatty acids, found that monounsaturates were neutral in their effect on LDL-C.9 Moderate-fat, moderate-carbohydrate diets could achieve LDL-C reductions equivalent to high-carbohydrate, low-fat diets.20 As the monounsaturated fat findings were replicated, later versions of the American Heart Association dietary guidelines appropriately focused on diets for reducing LDL-C via restrictions in saturated fat, rather than total fat.

An additional consequence to fat restricted diets is their propensity to increase fasting triglycerides (TG) and reduce high-density lipoprotein cholesterol (HDL-C). Within the range of total fat (18% to 40% kcal) evaluated in controlled feeding studies, there is a linear dose-response relationship between total fat content of the diet and the changes in HDL-C and TG.4 Weighted least-squares regression analysis revealed that for every 5% decrease in total fat, HDL-C levels would be expected to decrease by 2.2% and TG levels would be expected to increase 6%. Levels of Lp(a), a lesser studied atherogenic lipoprotein, also increase in a stepwise manner as levels of dietary total and saturated fat are reduced.21,22

Results from several recent clinical trials are highlighted in Table 1. The reduction in total fat in the recently published Women’s Health Initiative would have been expected to elicit ±4.4% reduction in HDL-C and ±12% increase in TG levels. The actual changes observed in this study were a 1.2% decrease in HDL-C and a 0.7% increase in TG levels.23 The investigators attribute this more favorable lipid change to the quality of the carbohydrate emphasized in the diet (fruits, vegetables, and whole grains). Similarly, in the OmniHeart Trial, a diet rich in carbohydrates resulted in only a modest decrease in HDL-C (~2.8%) and a negligible increase in TG (0.1%), compared with baseline.24 In the OmniHeart Trial, 3 diets were compared: high-carbohydrate, high-protein, and high-unsaturated-fat. As expected, the high-carbohydrate diet was associated with higher TG than the other diets; however, the high-protein diet resulted in a lower HDL-C than the high-carbohydrate diet. In the Portfolio Diet Study25 TG were decreased (~16.8 mg/dL) and the HDL-C decrease was blunted (~3.1 mg/dL), compared with the reduction observed on the control diet. The TG response observed in the DASH diet study (+3.5%) was much less than would have been predicted (+12%) given the 10% reduction in total fat, compared with the control diet. In these 5 newer trials, high-carbohydrate diets continued to improve the TC/HDL-C ratio, as observed in earlier trials. Thus, new data consistently support the current range for carbohydrate intake.

Enter the Metabolic Syndrome
Approximately 47 million US residents have the metabolic syndrome (MetS). It is estimated that the MetS will soon surpass cigarette smoking as the number one risk factor for cardiovascular disease risk in the US population. The National Cholesterol Education Adult Treatment Panel III defined the MetS as the presence of three or more of the following 5 factors: (1) abdominal obesity (waist circumference >102 cm for males and >99 cm for females); (2) elevated triglycerides >150 mg/dL; (3) low HDL-C (<40 mg/dL for men and <50 mg/dL for women); and (4) hypertension (>130/85 mm Hg for SBP/DBP) and elevated fasting glucose (>110 mg/dL). Lipid abnormalities that are present in the MetS but not included in its definition are small, dense LDL particles. Often referred to as the “lipid triad” (high triglycerides, low HDL-C and small, dense LDL particles) these lipid abnormalities are associated with elevated blood pressure, insulin resistance, and a prothrombotic state.

### TABLE 1. Recent Clinical Trials Evaluating Reduced-Fat/High-Carbohydrate Diets

<table>
<thead>
<tr>
<th>Study</th>
<th>Study Design</th>
<th>Length of Intervention</th>
<th>Subject Population</th>
<th>Diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women’s Health Initiative Trial (2006)23</td>
<td>Randomized, controlled free-living study</td>
<td>Mean follow-up: 8.1 year; CVD risk markers assessed at 3 years</td>
<td>48,835 postmenopausal women</td>
<td>Reduction in total fat (&lt;20% en) rich in fruits, vegetables and whole grains</td>
</tr>
<tr>
<td>OmniHeart Trial (2005)24</td>
<td>Randomized, crossover, controlled feeding study</td>
<td>Three 6-week feeding periods</td>
<td>164 men and women with pre-HTN or stage I HTN</td>
<td>Diet rich in carbohydrates</td>
</tr>
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<td>Three 6-week feeding periods</td>
<td>164 men and women with pre-HTN or stage I HTN</td>
<td>Diet rich in carbohydrates</td>
</tr>
<tr>
<td>Portfolio Diet Study (2003)25</td>
<td>Randomized, controlled feeding study</td>
<td>1 month</td>
<td>46 men and women with elevated cholesterol</td>
<td>Low-fat, high-fiber vegetarian diet</td>
</tr>
<tr>
<td>DASH diet Trial (2001)27</td>
<td>Randomized, controlled free-living study</td>
<td>8 weeks</td>
<td>436 men and women with pre-HTN or stage I HTN</td>
<td>Low-fat diet rich in fruits, vegetables, whole grains and low-fat dairy</td>
</tr>
</tbody>
</table>

CHO indicates dietary carbohydrate; HDL, high-density lipoproteins; HTN, hypertension; LDL, low-density lipoproteins; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids; TC, total cholesterol; TG, triglycerides.

*P<0.0001 when compared to control diet; †P<0.005 when compared to control diet; ‡P<0.05 when compared to control diet; §P<0.001 when compared to carbohydrate-rich diet; ¶P<0.05 when compared to carbohydrate-rich diet; ||P<0.001 when compared to protein-rich diet; **P<0.05 when compared to protein-rich diet.

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Aside from lowering LDL-C, diets enriched in carbohydrate are expected to have 2 adverse effects in patients with MetS.26 First, high carbohydrate intakes require higher insulin levels for postprandial metabolism. In the insulin-resistant state of MetS, the high carbohydrate load will raise postprandial glucose levels and in some patients raise fasting glucose levels if the carbohydrate load exceeds insulin secretion capacity. Second, if patients with MetS can compensate with greater insulin secretion, the increased magnitude of hyperinsulinemia would be expected to worsen the TG/HDL-C abnormalities already present in MetS.26

Feeding studies support this hypothesis. Carbohydrate-enriched diets have been shown to induce atherogenic dyslipidemia,27 which is characterized by small dense LDL particles, high TG levels, and low HDL-C levels. An analysis of several short-term feeding studies indicate that across a wide range of dietary carbohydrate (40% to 80% kcal) and fat (5% to 45% kcal) intake there is a strong linear relationship (r=0.93; P<0.0001) between the prevalence of LDL phenotype B (small dense LDL) and the percentage of calories from dietary carbohydrates.28 This dyslipidemia appears more pronounced in sedentary, overweight or obese populations.29

**Not All Carbohydrates Are Created Equal**

As with dietary fat, all carbohydrates are not chemically equivalent. Carbohydrates can be classified into the broad categories of simple or complex carbohydrates. Simple carbohydrates can be further broken-down into monosaccharides (eg, glucose and fructose) and disaccharides (eg, sucrose and lactose). Complex carbohydrates are either starch or fiber; fiber has several possible categorizations (eg, soluble versus insoluble, nondigestible versus lignan versus functional fiber provided from nondietary sources). Thus, the term carbohydrate describes a diverse macronutrient and recommendations regarding total carbohydrate intake reveal little about how the recommendations should be implemented using foods. Because processed foods containing added sugars are replacing other foods in the American diet, simple carbohydrate intake has increased to a greater extent than complex carbohydrates in the diet and now provides nearly one-third of total dietary carbohydrate intake (Figure).30

The Institute of Medicine of the National Academies has been acutely aware of differences within the carbohydrate subset and has set additional guidelines for fiber and added sugar at 14 g per 1000 kcal and 25% or less of energy from added sugars.4,31 In addition, practical advice identifies a category of “discretionary calories” (13% of intake), which could be consumed as added sugars.5

**Simple Carbohydrates and Added Sugars: Fructose Versus Glucose**

Simple carbohydrates, or sugars, are not all equivalent in their biologic effects. Sucrose, the carbohydrate found in table sugar, is sugar easily refined from sugar beets and sugar cane. As a disaccharide composed of fructose and glucose, sucrose has been used as a sweetener for centuries and until the 1990s was the major source of per capita sweetener in the American diet. Fructose is a monosaccharide named for the sweetener found in fruit. In the 1970s an inexpensive process was developed to convert cornstarch into high-fructose corn syrup. This syrup is a mixture of fructose and glucose monosaccharides. With the introduction of high-fructose corn syrup, American diets have become sweeter. Specifically, per capita consumption of sugar in the United States is 46.4 pounds, compared with 55.7 pounds for corn sweeteners (high-fructose corn syrup, glucose syrup, and dextrose). Food products which contribute the most to this increase in simple sugar consumption are grain products, including cakes, cookies, and cereals at 42.3%, and sugar, sweets, and beverages at 38.2% of total sugar consumption.32 According to data from the US Department of Agriculture’s Continuing Survey of Food Intakes of Individuals (CSFII) 1994 to 1996 and 1998, added sugars currently contribute 24% to 30% of dietary carbohydrates; the contribution of added sugar intake from fructose per se is estimated at 7% to 8% of calories, whereas fructose consumption from both added sugars and naturally occurring sugars in fruit and fruit juices is ≈12% calories.33

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**TABLE 1. Continued**

<table>
<thead>
<tr>
<th>CHO*</th>
<th>Protein*</th>
<th>Total Fat*</th>
<th>SFA*</th>
<th>MUFA*</th>
<th>PUFA*</th>
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<th>LDL (mg/dL)</th>
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<tr>
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Fructose metabolism differs from that of sucrose and glucose, and consequently has different implications for health. The majority of fructose metabolism occurs in the hepatic cytoplasm, where it is phosphorylated by fructokinase to fructose-1-phosphate. Fructose-1-phosphate is then catalyzed by aldolase B to form dihydroxyacetone-3-phosphate and glyceraldehyde. These triose phosphates continue through the Emden-Meyerhof pathway yielding pyruvate, which enters the mitochondria for the citric acid cycle, or glycerol-3-phosphate, which provides the glycerol moiety for triacylglycerol synthesis. Compared with glucose metabolism, where citrate produced by mitochondria inhibits the enzyme phosphofructokinase, shutting off additional catabolism of glucose to fructose-1-phosphate, fructose catabolism proceeds without feedback regulation. Thus, when large quantities of fructose are consumed, the system can be flooded with intermediates including acetyl coenzyme A for lipogenesis and triglyceride synthesis. Clinical studies have demonstrated that fructose consumption results in substantial increases in lipogenesis compared with consumption of eu-caloric amounts of glucose.

Several studies have compared the effects of glucose versus fructose on lipid metabolism. Bantle et al. compared the effects of consuming 17% of calories from either fructose or glucose for 6 weeks. Plasma TG levels significantly increased in men ($P<0.001$) but not women ($P<0.72$) after the fructose period. LDL-C was higher at the 4-week time point during fructose feeding, but this difference was no longer apparent at 6 weeks. Havel et al. evaluated the effects of consuming a chronic diet of either 25% kcal as fructose or glucose on the postprandial triglyceride concentrations of overweight or obese women with normal triglycerides. Postprandial triglyceride concentration increased in the area under the curve over 14 hours in the subjects consuming the 25% fructose diet compared with their baseline complex carbohydrate diet. The magnitude of the increase was greater at 10 weeks on the fructose treatment compared with 2 weeks. At 10 weeks, apolipoprotein B levels increased 11.7+3.7% among subjects consuming 25% fructose but no such increase was observed on the glucose treatment. Postprandial triglycerides were not significantly altered from the baseline diet at 10 weeks for either glucose or fructose treatments.

In summary, simple carbohydrates are, by their namesake rapidly absorbed. The predominant effect on serum glucose and lipid metabolism depends on the type of simple carbohydrate and the insulin resistant state of the individual ingesting the food or beverage. Simple carbohydrates such as glucose may raise postprandial glucose in patients with MetS; high-fructose corn syrup may improve postprandial glucose at the expense of increasing postprandial triglycerides. Neither effect should be touted as an improvement in cardiovascular risk.

Complex Carbohydrates: A Focus on Fiber

In contrast to the deleterious effects of simple carbohydrates on lipid and glucose metabolism, dietary fiber has demonstrated consistent beneficial effects on lipid metabolism. Numerous studies have shown that diets rich in soluble fiber lower blood cholesterol levels, whereas diets rich in insoluble fiber have no effect. The major soluble fibers are β-glucan (found in oats, barley, and yeast), psyllium (found in husks of blonde psyllium seed), and pectin (found in fruit). Several properties of soluble fiber, viscosity, bile acid binding capacity, and potential cholesterol synthesis-inhibiting capacity after fermentation in the colon contribute to its cholesterol-lowering effect.

A meta-analysis of 8 studies reported that 10 g per day of psyllium reduced TC and LDL-C levels by 4% and 7%, respectively. Another meta-analysis of 67 controlled dietary studies found that for each gram of soluble fiber from oats, psyllium, pectin, or guar gum, TC concentrations decreased by 1.42, 1.10, 2.69, and 1.13 mg/dL, respectively. Similarly, LDL-C levels decreased by 1.23, 1.11, 1.96, and 1.20 mg/dL,
respectively, demonstrating comparable cholesterol-lowering effects of these soluble fibers. In a study of normolipidemic and normotensive subjects (n/H11005/53), an increase in dietary fiber intake (30.5 g/d total fiber and 4.11 g/d soluble fiber) significantly reduced LDL-C (12.8%), but did not affect TG or HDL-C levels.51 The addition of 3 or 6 g/d /H9252/-glucan from barley to a Step I diet (55% cholesterol, 16% protein, 31% total fat) has been shown to lower TC (4% and 9%, respectively) and LDL-C (13.8% and 17.4%, respectively) concentrations in mildly hypercholesterolemic men and women.52

Translating the disparate effects of simple versus complex carbohydrates, easily shown in experimental feeding studies, into dietary choices has been difficult because most foods contain both simple and complex carbohydrates. Two measures to combine the overall food effect, glycemic index (GI) and glycemic load (GL), have been proposed. The GI is defined as the area under the 2-hour glycemic curve after consumption of a food containing 50 grams of carbohydrate, divided by the area under the curve for a standard food (white bread or glucose).53,54 The GL is defined as the product of the GI of that food multiplied by its carbohydrate content. Clinical studies generally show that when the amount of carbohydrate is held constant, foods with a higher GI increase fasting TG levels.55 Low-GI diets also may protect against HDL-C–lowering associated with traditional high-carbohydrate diets.56 The large variability in response to specific foods and mixed meals composed of foods with different GI/GL has led to somewhat unwieldy consideration of this index in dietary recommendations. Simple food descriptions continue to predominate in dietary carbohydrate guidelines (eg, whole grains, no added sugars, etc).

**Carbohydrates and Weight Loss**

Weight loss is the metabolic consequence of reduced caloric intake. How dietary carbohydrate recommendations fit within weight loss programs depends on the style of the dietary change. Extreme diets, either very high in carbohydrates or very low in carbohydrates in which energy intake is similarly restricted, have been successful at achieving weight loss.57 For example, the Pritikin Program, a lifestyle intervention program that includes a high-complex-carbohydrate, high-fiber (35 to 40 g/1000 kcal), very low-fat (<10% kcal), and low-cholesterol (<25 mg/d) diet and exercise component, has been shown to reduce weight, TC, and TG by 5%, 21%, and 50%, respectively.58 Participants are satiated on high-fiber/low-energy density diets that are hypocaloric. In contrast, The Atkins Diet, a diet intervention program, produces a caloric restriction through the elimination of carbohydrate-containing foods such as bread, pasta, and fruit, can also produce weight loss. Participants report satiety from high protein meals; the ketotic effect of very low carbohydrate intakes also reduces hunger. Given the many weight loss diets, an important question is whether there is one weight loss program that is better than others, and does the carbohydrate content of the weight-loss diet predict its success?59

A recent study compared the 1-year effects of 4 weight loss diets (Atkins, Ornish, Weight Watchers, Zone) conducted in an outpatient, diet counseling setting.59 The weight loss diets varied appreciably in their carbohydrate content (high, Ornish; intermediate, Zone and Weight Watchers; very low, low, and very low).
Atkins); for the first few months participants followed their assigned intakes. However, throughout the full year there was a gradual drift in dietary intakes so that the 1-year macronutrient profiles of the four diet groups were not markedly different (39% to 48% kcal cholesterol; 31% to 39% kcal total fat), but rather similar to baseline values. After 12 months there was a very modest and similar weight loss (2 to 3 kg) in subjects following the different diets, with comparable decreases in LDL-C (range, 7.1 to 12.6 mg/dL) and the TC:HDL-C ratio observed. This study suggests that extremes in dietary intake are not sustainable over the long term. It also suggests that any diet program can achieve small but sustainable weight loss in motivated participants.

Practical Recommendations and Conclusions
Dietary recommendations suggest moderate amounts of both carbohydrate (45% to 65% kcal) and fat (20% to 35% kcal) in the diet. Dietary patterns within these ranges for carbohydrate and fat have been shown to promote weight loss, lower TG levels, maintain HDL-C levels, and promote maintenance of reduced body weight. In overweight patients, reduced-fat, high-carbohydrate diets have proven effective for weight loss, the prevention of type 2 diabetes, and MetS.

An important point to consider with all of these interventions is that the reduced-fat, high-carbohydrate diets tested were used in conjunction with other lifestyle changes, including dietary patterns emphasizing whole grains, fruits, and vegetables and lifestyle patterns increasing daily physical activity. If the American dietary pattern continues to incorporate additional processed foods and more high-fructose corn syrup-sweetened foods, carbohydrate intake could contribute to an increase in cardiovascular disease risk factors. Clinicians should not only follow the macronutrient recommendations but also pay attention to the food patterns that meet these recommendations. Recommendations for dietary carbohydrates are only met when fruits, vegetables, whole grains, and nonfat dairy products are included in the daily diet (Table 2).

Disclosures
None.

References


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