A New Look at Carbohydrate-Restricted Diets

Article *in* Nutrition Today · March 2013

DOI: 10.1097/NT.0b013e31828814eb

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A New Look at Carbohydrate-Restricted Diets

Separating Fact From Fiction

Jeff S. Volek, PhD, RD
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After 30 years of policy based on the Diet Heart (low-fat) Hypothesis, obesity and type 2 diabetes are now an epidemic. Recently published research seriously questions the dangers of dietary saturated fats and also any potential health benefits of a low-fat diet for the general population. In the context of these seismic changes in evidence-based nutrition, it is time to revisit our perspective of carbohydrate-restricted diets, not only for weight loss but also for the long-term management of conditions associated with insulin resistance. In randomized trials comparing high-carbohydrate versus low-carbohydrate diets, a well-formulated low-carbohydrate diet reduced the absolute concentration of saturated fat in serum lipids. This apparent paradox is explained by the accelerated ß-oxidation of saturated fatty acids when humans are adapted to a ketogenic diet. Given that this keto-adapted state is also associated with no reduction in physical performance and reduced inflammation, carbohydrate-restricted diets may play an important role in the prevention and treatment of diseases of insulin resistance such as metabolic syndrome and type 2 diabetes. Nutr Today. 2013;48(2):E1–E7

REDEFINING AVERAGE

Thirty years ago, less than half of adults in the United States were classified as overweight or obese. Since then, we contend that Americans have been part of a massive experiment to test the effects of a low-fat diet. Few would have predicted the outcome; the average person is now overweight. Nearly 3 of 4 individuals currently have a body mass index of 25 kg/m^2 or greater, and 1 in 3 adults are obese (body mass index ≥30 kg/m^2).[1] One irrefutable conclusion from our national experience is that the one-size-fits-all "public health approach" that encouraged people to eat less fat has been unsuccessful for many, if not most, of us. The major reason is that most people replaced those fat calories with an even greater amount of carbohydrate, thereby increasing their total caloric intake. The fact that only a relatively small subset of adults has been able to maintain a healthy weight in the context of current low-fat dietary guidelines implies that something approaching most Americans may be metabolically and genetically programmed to benefit from alternative approaches.

Despite several lines of evidence pointing to the need to personalize diet approaches based on genetic and nongenetic factors, our current understanding of how these factors interact to produce favorable health outcomes is rudimentary. Interestingly, for most people, the ability to respond favorably to a low-carbohydrate diet appears to be imprinted in their genes.[2] With significant advancements in nutritional sciences, coupled with new technologies in modern genetic and biomarker assessment, personalized healthcare is primed to become a common practice over the next decade. However, although DNA-guided dietary advice may be on the horizon, our burgeoning epidemic of obesity dictates that we reexamine current doctrine and take evidence-based action now.

REEXAMINING THE ROLE OF CARBOHYDRATE

Looking at our dietary patterns over the last few decades, the most salient change associated with the obesity and diabetes epidemic has been the marked increase in dietary carbohydrate consumption. Over the last few decades, Americans have increased mean daily energy intake by about 200 kcal, due exclusively to an increase in carbohydrate, whereas protein remained the same and fat actually decreased slightly.[3] Should we be concerned about ingesting this additional carbohydrate? Although mere association does not prove causality, there are mechanistic reasons why increased carbohydrate intake, particularly of simple sugars, can lead to net body fat deposition.

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Dr Volek's work on low-carbohydrate diets has been supported by funds from the University of Connecticut Graduate School, the Health Disparity EXPORT Center at the University of Connecticut, US Department of Agriculture Hatch Funds, the Dr. Robert C. Atkins Foundation, and the Egg Nutrition Center.

Drs Volek and Phinney were coauthors on the *New Atkins for a New You*, released in March 2010, and the Art and Science of Low Carbohydrate Living, released in May 2011.

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DOI: 10.1097/NT.0b013e31828814eb

In the body's hierarchy of fuel selection, glucose is oxidized in preference to fat. This is because the human body has a limited capacity to store carbohydrate as glycogen (~300–500 g) but a virtually unlimited ability to store fat. The conversion of carbohydrate into fat is called de novo lipogenesis (DNL). Because total body free sugar in a healthy adult is at most 20 g, a meal containing 100 g of carbohydrate must be rapidly disposed of by some combination of direct oxidation, glycogen deposition, or DNL to avoid acute hyperglycemia. Thus, it comes as no surprise that excess carbohydrate intake increases lipogenesis in healthy individuals.[4] Even a single meal of sugar, especially fructose, immediately elevates rates of lipogenesis and increases serum triglyceride levels.[5]

One's ability to metabolize and partition carbohydrate calories is further dependent on the level of insulin resistance. In normal-weight, insulin-sensitive men who consumed 2 meals containing 55% carbohydrate, most of the carbohydrate was taken up by skeletal muscle.[6] In contrast, men showing early signs of insulin resistance diverted significantly more of the ingested carbohydrate into hepatic DNL, resulting in >2 fold increases in hepatic triglyceride synthesis and plasma triglycerides, and lower plasma HDL. These studies provide evidence that consumption of too much carbohydrate is readily converted to fat, leading to dyslipidemia in otherwise healthy individuals with early signs of insulin resistance.

Whereas metabolism of a high-carbohydrate diet poses problems for some individuals (ie, those who readily convert carbohydrate to fat), it has been known for at least 60 years that fasting or removal of dietary carbohydrate results in a virtual absence of DNL.[7] However, whereas total fasting is clearly not a sustainable approach, a well-formulated low-carbohydrate diet is a viable long-term strategy. The normal response to fasting (decreased glucose and insulin, increased availability of fatty acids and ketones, and accelerated breakdown of stored body fat) is mimicked by a very-low-carbohydrate diet.[8,9] Even small decreases in insulin are associated with large increases in fat breakdown and fat oxidation.[10] A key point is that the increased use of lipid fuels characteristic of fasting is not caused by restriction of calories per se but rather by a reduction in carbohydrate. In other words, many of the positive effects of fasting in respect to upregulating the body's ability to mobilize and use fat for fuel are driven primarily by a reduction in dietary carbohydrate.

The authors claim that low-carbohydrate diets are grounded in well-established biochemical principles, and that many old and new studies, demonstrate positive metabolic effects.

LOW-CARBOHYDRATE DIETS POSSIBLY BENEFIT METABOLIC SYNDROME (INSULIN RESISTANCE SYNDROME)

The prevalence of diabetes has increased in parallel with escalating obesity. In 2007, an estimated 23.5 million (10.7%) adults in the United States had diabetes, and approximately 3 times that number of people have prediabetes based on increased fasting glucose level, an early sign of insulin resistance. *Metabolic syndrome* is the commonly used term to describe an insulin-resistant state that manifests in varying ways such as excess adiposity, dyslipidemia (high triglycerides, low HDL cholesterol), hypertension, high blood glucose and insulin, inflammation, and vascular dysfunction. It is important to note, however, that whereas metabolic syndrome carries a high risk of leading to diabetes, progression from early signs of insulin resistance to prediabetes to diabetes is neither inevitable nor irreversible.

When looked at from a purely functional perspective, metabolic syndrome, insulin resistance, diabetes, and even many forms of obesity are all manifestations of carbohydrate intolerance. Consistent with the classic paradigm for the dietary management of gluten, lactose, or fructose intolerances, it therefore follows that carbohydrate restriction would be an intuitive and rational approach to improvement of glycemic and metabolic control in the presence of insulin resistance. Many research teams, including significant work by the authors, have repeatedly shown that low-carbohydrate diets are more likely than low-fat diets to effect global improvement in markers associated with insulin resistance. These include improvements in plasma glucose, insulin, triglycerides, HDL cholesterol, low-density lipoprotein (LDL) particle size, inflammatory markers, and vascular function.[11,12]

UPDATED PERSPECTIVE ON SFA

A barrier for some people considering low-carbohydrate diets is the perception that increasing saturated fat (SFA) intake poses a health risk. Current US dietary guidelines encourage Americans to limit SFA to less than 7% of energy. This chain of reasoning, often referred to as the Diet-Heart Hypothesis, has recently been the topic of increasing debate.[13] A recent test of this hypothesis, the Women's Health Initiative, using a low fat, low saturated fat diet did not result in significant decreases in heart disease, diabetes or cancer risk.[14] Furthermore, a recent meta-analysis has shown that dietary SFA is not a risk factor for cardiovascular disease[15] and that replacement of dietary fat by carbohydrate (exactly what many Americans have done) may actually increase risk of coronary events.[16]

The extent to which plasma SFA reflects dietary SFA consumption is not straightforward and is significantly affected by the presence of carbohydrate. Counterintuitively, previous studies have reported lower plasma levels of SFA in

response to diets that contained 2- to 3-fold greater intake of SFA but were lower in carbohydrate.[17,18] In 2 recent studies, we assessed fatty acid composition in plasma lipid fractions from adults consuming either a carbohydrate-restricted diet or a low-fat diet. In the first study, men and women with metabolic syndrome consumed a carbohydrate-restricted diet or a similarly hypocaloric low-fat diet.[19] There was a significantly greater reduction in plasma SFA levels in response to a low-carbohydrate diet, despite a 3-fold greater intake of dietary SFA.

We further extended these observations in highly controlled feeding experiments designed to maintain body weight. Weight-stable men were fed two 6-week low-carbohydrate diets (12% carbohydrate) that emphasized SFA (86 g/d SFA) or unsaturated fat (UFA) (47 g SFA/d).[19] All foods were provided to the subjects. The UFA low-carbohydrate diet decreased the proportion of saturates in plasma triglycerides (25.8 ± 2.9 wt%) compared with baseline (29.2 ± 4.1 wt%). Plasma triglyceride SFA, however, remained unchanged in the SFA low-carbohydrate diet (31.3 ± 3.4 wt%) despite a doubling in SFA intake (Figure 1).

However, because plasma triglycerides were markedly reduced with both the UFA and SFA diets, the absolute SFA concentration in plasma triglycerides was decreased by ~50% (Figure 1, right panel), similar to the 57% decrease we observed in response to a hypocaloric low-carbohydrate diet.[19] These results can best be explained by the metabolic adaptations induced by carbohydrate restriction,[9,11,12] notably less inhibition of fat oxidation by insulin, and thus, rapid β-oxidation of saturated fatty acids.

The plasma lipid fraction most responsive to carbohydrate restriction in our studies was triglyceride, which is consistent with its role as the body's primary circulating energy supply when carbohydrate intake and whole-body glucose flux are reduced. And paradoxically, this explains the observation that higher incorporation of SFA into very-LDL triglyceride is correlated with insulin resistance and adiposity,[20] where the increased saturated fatty acids are not reflecting dietary fat intake but instead accelerated hepatic DNL. This new paradigm, that dietary carbohydrate has a major influence on the metabolic processing of SFA and the consequent cardiometabolic risk profile, is shown in Figure 2.

The authors claim that a low-carbohydrate diet reduces SFA levels irrespective of dietary SFA intake.

HISTORICAL INSIGHTS PERTINENT TO THE SAFETY AND EFFICACY OF CARBOHYDRATE RESTRICTION

This new perspective on the benign nature of dietary SFA in the context of a low-carbohydrate intake helps explain how individuals in inland aboriginal hunting and herding cultures were able to eat diets rich in SFAs and nonetheless remain healthy. Published observations of food preferences of precontact aboriginal hunters and herders indicate that their energy intakes consisted mostly of fat with protein in moderation (15–30 energy%).[21–23]

Interestingly, both Catlin[21] and Orr and Gilks[23] commented on the physical proportions of their respective subjects of observation. In the case of Catlin, a painter, he noted that the Native American cultures that lived by hunting bison on the Great Plains were much taller (typically between 6 and 7 ft tall) than contemporary Europeans in North America, and with no evidence of corpulence. In the comparison by Orr and Gilks of the Masai versus the Kikuku living in central Kenya, their careful anthropometrics demonstrated that the Masai men (who eschewed all vegetable-origin foods) were 5 in. taller, on average, than the Kikuku men, who lived in adjacent regions by subsistence farming. In the authors' words: "the Masai are typically larger in the chest and smaller in the abdomen than the Kikuku."

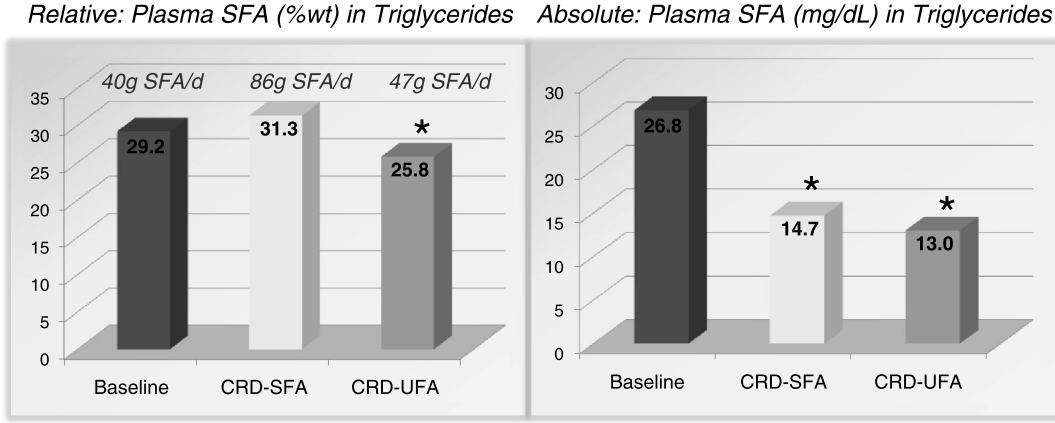

FIGURE 1. Plasma saturated fat (SFA) response at baseline (low-fat diet) and after two 6-week carbohydrate-restricted diets (CRD) that emphasized SFA (CRD-SFA) and unsaturated fat (CRD-UFA).

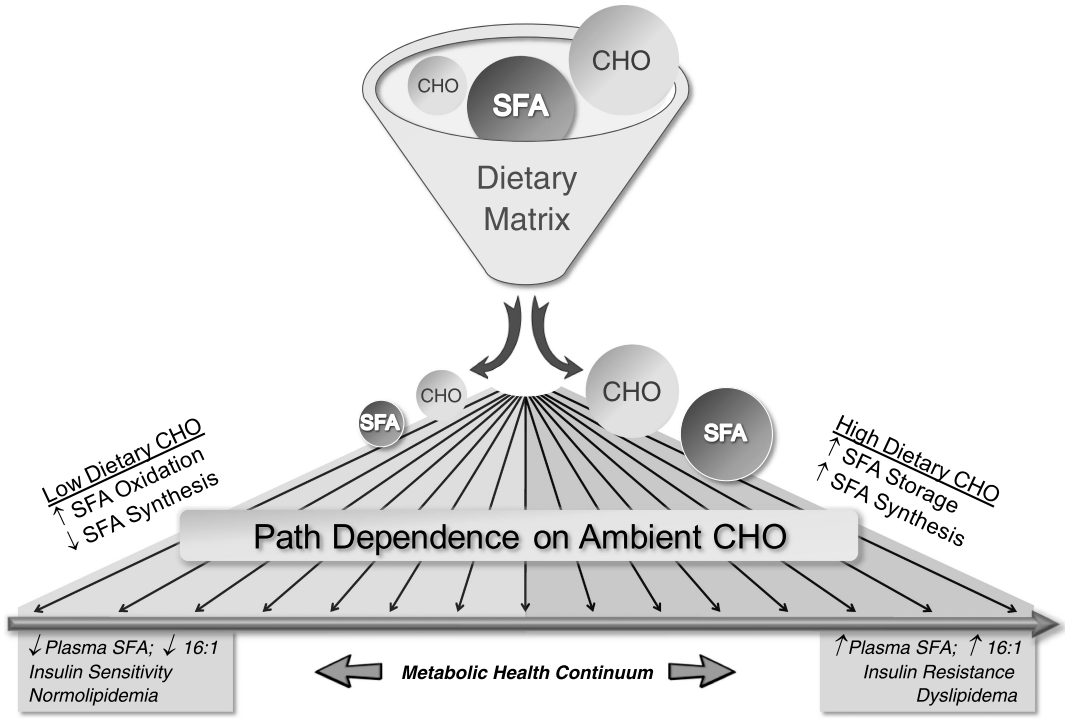

FIGURE 2. Paradigm depicting the importance of dietary carbohydrate on metabolic processing of saturated fat (SFA) and consequent metabolic health. The major point is that SFA, and the response to eggs, has a totally different metabolic behavior when consumed in the context of a low-carbohydrate diet. Abbreviation: CHO, carbohydrate.

In addition to greater height and musculature, Orr and Gilks demonstrated significantly higher strength values among the Masai using hand dynamometry. These are not scientific studies, but these accounts do provide evidence that low-carbohydrate diets are compatible with general health and the ability to perform physical activity. We[24] have also assessed endurance performance in bicycle racers adapted to a very-low-carbohydrate, adequate-calorie diet for 4 weeks and found no reduction in either peak aerobic power or endurance time at 65% of peak power. Taken in combination, these observations throw into question the common assumption that a diet rich in fat necessarily causes obesity and impaired physical function.

SUSTAINABILITY

Short-term use of a low-carbohydrate diet is often effective at inducing major weight loss and improvements in metabolic health, especially in those who have insulin resistance and metabolic syndrome. However, returning to one's habitual level of carbohydrate intake will inevitably result in reversal of these benefits. Thus, for long-term success, the patient/client must be able to sustain a level of carbohydrate restriction that is compatible with his/her unique metabolism. A mistake too often made is that carbohydrates are reintroduced at a level that exceeds a person's tolerance. The composition of the calories that must be increased when one progresses from a weight loss to a weight maintenance phase is critical. Carbohydrates may be increased slightly based on individual tolerance, but the majority of calories will need to come from fat.

In most longer term studies of low-carbohydrate diets, however, rather than encouraging subjects to consume more fat in the later phases of the diet, they often reintroduce high-carbohydrate foods. Thus, it is not surprising that in randomized clinical trials, weight loss at 6 months is higher in groups assigned to a low-carbohydrate diet than in those assigned to a low-fat diet, but the effect is diminished at 12 months.[25] Nevertheless, weight loss is at least as good and some cardiovascular risk markers improve to a greater extent, especially in subjects with insulin resistance.[26]

There have been relatively few published studies that have successfully encouraged subjects to adhere to a carbohydrate intake of less than 10% of energy intake long-term. One was the classic Bellevue study by McClellan and Dubois,[27] in which 2 Arctic explorers were sequestered in a metabolic ward for a number of months and then closely monitored as outpatients for a total of 12 months each. Throughout this period, they ate a diet patterned after that of the precontact Inuit among whom they had lived in the Arctic, and both maintained their health and function for the duration of the study. Of particular note, the diet they ate (which was meticulously analyzed and reported) consisted of 15% protein (ie, not "high protein"), approximately 80% as fat, and less than 5% from all forms of carbohydrate.

TABLE Example Daily Meal Plan of a 2500 kcal Very-Low-Carbohydrate Diet
Breakfast (scrambled eggs with sides of spinach and sausage)
Scrambled eggs: 2 large + 1 tbsp palm oil
Mozzarella cheese: 1 oz
Pork sausage: 2 links (48 g)
Chopped frozen spinach, boiled: 3/4 cup (142.5 g) + 1.5 tbsp butter
Snack
1/2 Avocado: 67 g
Swiss cheese: 2 oz (56 g)
Lunch (broiled salmon and a side salad)
Broiled Atlantic salmon: 4 oz + 1 tbsp butter
Side salad: mixed baby greens: 2.5 cups
Diced tomatoes: 1/4 cup
Chopped onion: 1/8 cup
Feta cheese: 1 oz
Black and green olives: 4 each
Blue cheese dressing: 1.5 tbsp
Snack
Peanuts, oil-roasted: 1 oz
Hood calorie countdown milk: 1/2 cup
Dinner (sirloin with sautéed mushrooms and cauliflower ''mashed potatoes)
Beef sirloin tips: 3 oz
Olive oil: 1.5 tbsp
Sautéed mushrooms: 1/4 cup
Olive oil cooking spray
Cauliflower ''mashed potatoes'': boiled cauliflower—1 cup + shredded cheddar cheese—1 oz
Butter: 1 tbsp
Sugar-free jello: 1/2 cup (121 g)
Protein: 134 g
Carbohydrates: 42 g, Fiber 20 g
Fat: 204 g
Cholesterol: 853 mg
SFA: 81 g
MUFA: 78 g
PUFA: 28 g
Abbreviations: SFA, saturated fat; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.

More recently, a clinical practice in Kuwait has adopted a low-carbohydrate, moderate-protein, high-fat diet to deal with the dramatically increased incidence of obesity and type 2 diabetes in that region.[28] This publication reports a cohort of 66 subjects (35 with type 2 diabetes) who were instructed to maintain total daily carbohydrate intake under 50 g/d, 49 of whom achieved a mean weight loss of 27 kg in a year, with dramatic improvements in dyslipidemia and normalization of blood glucose among the diabetics. Given that intensive pharmaceutical management of type 2 diabetes to achieve

normoglycemia consistently leads to weight gain (as opposed to marked weight loss in this study), it is hard to believe that any potential (but as yet undemonstrated) risks of long-term carbohydrate restriction outweigh the benefits of this dietary approach in this group of patients.

AUTHORS' CONTENTIONS

A detailed discussion of the appropriate composition of a well-formulated low-carbohydrate diet, both for weight loss and then for long-term maintenance, has been addressed in 2 recent books we coauthored: *The New Atkins for a New You*[29] and *The Art and Science of Low Carbohydrate Living*.[30] The former is written for the lay public, whereas the latter is directed at healthcare professionals. The degree of carbohydrate restriction will vary from person to person, but for those with carbohydrate intolerance, this will likely not exceed 100 g/d and may be as low as 30 g/d in the most carbohydrate-intolerant individuals. In brief, a well-formulated low-carbohydrate diet is moderate (not high) in protein; includes daily consumption of a variety of vegetables; is attentive to mineral management, including added sodium in the form of broth/bouillon to compensate for the natriuresis of carbohydrate restriction; allows the addition of fat as the diet progresses from weight loss to maintenance; and limits high amounts of polyunsaturated fat while emphasizing monounsaturated and SFAs for fuel. Following these guidelines, a wide range of nutrient-rich satisfying meals can be created at very low carbohydrate intakes (Table).

CONCLUSIONS

Our current epidemic of obesity and its association with increased morbidity and mortality underscore the need for a more sophisticated approach to dietary policy. Among the many misunderstandings about low-carbohydrate diets, some of the most persistent are that they are dangerously high in protein, that the amount of SFA consumed is dangerous, and that carbohydrate deprivation reduces well-being and physical function. All of these are manifestly incorrect. As developed by aboriginal cultures over thousands of years, dietary fat sources were sought out and treasured, protein was eaten in moderation, and gathered foods were, at most, a minor energy source (albeit potentially important sources of vitamins and minerals). Recent research showing that humans rapidly clear SFAs and have reduced inflammation biomarkers when consuming well-formulated low-carbohydrate diets should encourage us to reexamine the therapeutic value of carbohydrate restriction. And because this research has also resolved some of the apparent paradoxes that have impeded the safe and sustained use of such diets, the immediate opportunity is to harness these benefits in the management of insulin resistance.

What does all this mean for health professionals? It means that we should have an open mind about the use of low-carbohydrate diets in the management of obesity, metabolic syndrome, insulin resistance, dyslipidemia, type 2 diabetes, and potentially other clinical conditions. Within the population, there are widely varying levels of carbohydrate tolerance, making it challenging to define a precise level of carbohydrate intake that is ideal for all individuals. The presence of any of the above conditions is a sign that there may be underlying carbohydrate intolerance, in which case health professionals should consider being supportive rather than dismissive of low-carbohydrate diets, seeking out ways to help and support the patient/client achieve and sustain a healthy diet low in carbohydrate.

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Editor's Note

The article "A New Look at Carbohydrate-Restricted Diets" presents some controversial views by the authors of 2 popular books supporting such regimens. There is merit in continuing to examine the evidence for and against low-carbohydrate diets. However, *Nutrition Today* readers need to keep in mind that the contention that Americans have been part of a massive experiment to test the effects of a low-fat diet is not apparent in national population-based data published in the Data Brief of the National Center for Health Statistics (no. 49, November 2010) entitled "Trends in Intake of Energy and Macronutrients in Adults From 1999–2000 Through 2007–2008." Those data show that there has been an increase in caloric intake among Americans and, if anything, a decrease in carbohydrate intake over this period when it is expressed as a percentage of calories. *Nutrition Today* does not endorse widespread implementation of low-carbohydrate diets, nor does it advocate that dietary recommendations should be changed in light of the authors' contentions. It urges readers to continue to evaluate the evidence.